**SUDBURY AREA RISK ASSESSMENT**

# VOLUME II – CHAPTER 4: DETAILED HUMAN HEALTH RISK ASSESSMENT METHODOLOGY

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# 4.0 DETAILED HUMAN HEALTH RISK ASSESSMENT METHODOLOGY

The detailed HHRA was conducted using the data from Phases 1 and 2, and followed the four major steps of the HHRA framework: i) problem formulation; ii) exposure assessment; iii) hazard assessment; and, iv) risk characterization. The problem formulation step (Phase 1) was previously discussed in detail in Chapter 2, while the additional sampling and analytical work conducted to fill identified data gaps was outlined in Chapter 3 of this volume.

This chapter will provide a detailed discussion of the estimated exposures and resulting risks to human health under each scenario evaluated as part of the HHRA. This chapter is comprised of the remaining three steps of the HHRA framework including the exposure assessment, hazard assessment and risk characterization.

## 4.1 Exposure Assessment

The exposure assessment evaluates data related to all chemicals, receptors and exposure pathways identified during the problem formulation phase of the HHRA using a multimedia approach. The multimedia approach takes into account all potential exposure to the COC from the different sources or media (*i.e.,* soil, air, dust, water, food, *etc*.) which typical Sudbury residents could come in contact with as part of their daily activities.

The primary objective of the exposure assessment is to predict, using site-specific data and a series of conservative assumptions, the rate of exposure (*i.e.,* the quantity of chemical and the rate at which that quantity is received) of the selected receptors to the COC *via* the various exposure scenarios and pathways identified in the problem formulation step. The rate of exposure to chemicals from the various pathways is usually expressed as the amount of chemical taken in per body weight per unit time (*e.g.,* µg chemical/kg body weight/day).

The degree of exposure of receptors to chemicals in the environment depends on the interactions of a number of parameters, including:

- The concentrations of chemicals in various environmental media;
- The physical-chemical characteristics of the COC, which affect their environmental fate and transport and determine such factors as efficiency of absorption into the body of a given external exposure;

- The influence of site-specific environmental characteristics, such as geology, soil type, topography, hydrology, hydrogeology, local meteorology and climatology *etc.* on a chemical's behaviour within environmental media; and,
- The physiological and behavioural characteristics of the receptors (*e.g.,* respiration rate, soils/dust intake, time spent at various activities and in different areas).

The rate of exposure to the COC available to residents of the GSA was evaluated through the estimation of an exposure point concentration (EPC) for each media type. Based upon U.S. EPA (2004a) guidance, the 95% upper confidence limit of the mean (*i.e.*, 95% UCLM) was used to estimate the reasonable maximum exposure (RME) point concentration. The arithmetic mean of the data set was used to estimate the central tendency estimate (CTE) point concentration. This value was calculated for each COC using ProUCL software developed by the U.S. EPA (2004b). ProUCL tests the data set for normality, lognormality, and gamma distributions using parametric and non-parametric methods to calculate a conservative and stable 95% UCLM (U.S. EPA, 2004b). ProUCL output summaries for all 95% UCLM values calculated for each exposure media are provided on the CDs accompanying this report.

For the purpose of statistical analyses of the data, any negative concentration or zero value was set equal to half the detection limit. Any value measured at, or below, the detection limit was also set equal to ½ the detection limit. Applying half the detection limit to the negative concentration and zero values was considered to be a conservative method to evaluate the data set provided. The uncertainties related to this assumption are discussed further in Chapter 7 of this volume.

### 4.1.1 Media Concentration Data Selected for Use in the HHRA

The following section provides a summary of the various media concentration data selected for use in the current HHRA.

#### 4.1.1.1 Surface Soil Concentrations

As the first phase of the Sudbury Soils Study, a comprehensive soil sampling program was undertaken in 2001 to collect more recent soil concentration data for the Sudbury region and to expand the existing soil database for Sudbury in terms of spatial coverage (*i.e.*, geographic area). The soil sampling program was conducted by three investigation teams and covered approximately a 40,000 $km^2$ area, including the City of Greater Sudbury (see Figure 4-1). Briefly, the MOE conducted an urban soils survey including the collection of over 6,000 soil samples (originals and replicates) from schools, daycares, parks, beaches and residential properties across the GSA (MOE, 2003). Laurentian University's Centre for Environmental

Monitoring (CEM) collected soil samples in more rural and remote areas of Sudbury to determine the spatial extent of the smelter "footprint" and to determine background concentrations of metals in the GSA (CEM, 2004). Finally, Golder Associates completed the Falconbridge Soil Survey in which soil samples were collected from residential properties and some municipal and crown lands in and around surrounding the Town of Falconbridge (Golder Associates, 2001). Each team used the same sampling design and approach to collect the samples from their respective zones. The current section is only intended to provide the reader with an overview summary of the data used in the current assessment. More details regarding these three surveys can be found in the *Summary Report: 2001 Sudbury Soils Data* (SARA, 2004) and in Volume I of the Sudbury Soils Study.

This comprehensive database of soil concentrations provided the data foundation of the HHRA, and formed the basis of screening and selection of chemicals of concern (COC) and communities of interest (COI) for the current assessment. For the purposes of the HHRA, the soils database was screened using several criteria to evaluate exposure scenarios and soil concentrations most relevant to the exposure of residents of the GSA. Initially, more than 2,100 surface soil samples were extracted from the database, which included the original and replicate samples for each chemical of concern (COC) in each community of interest (COI).

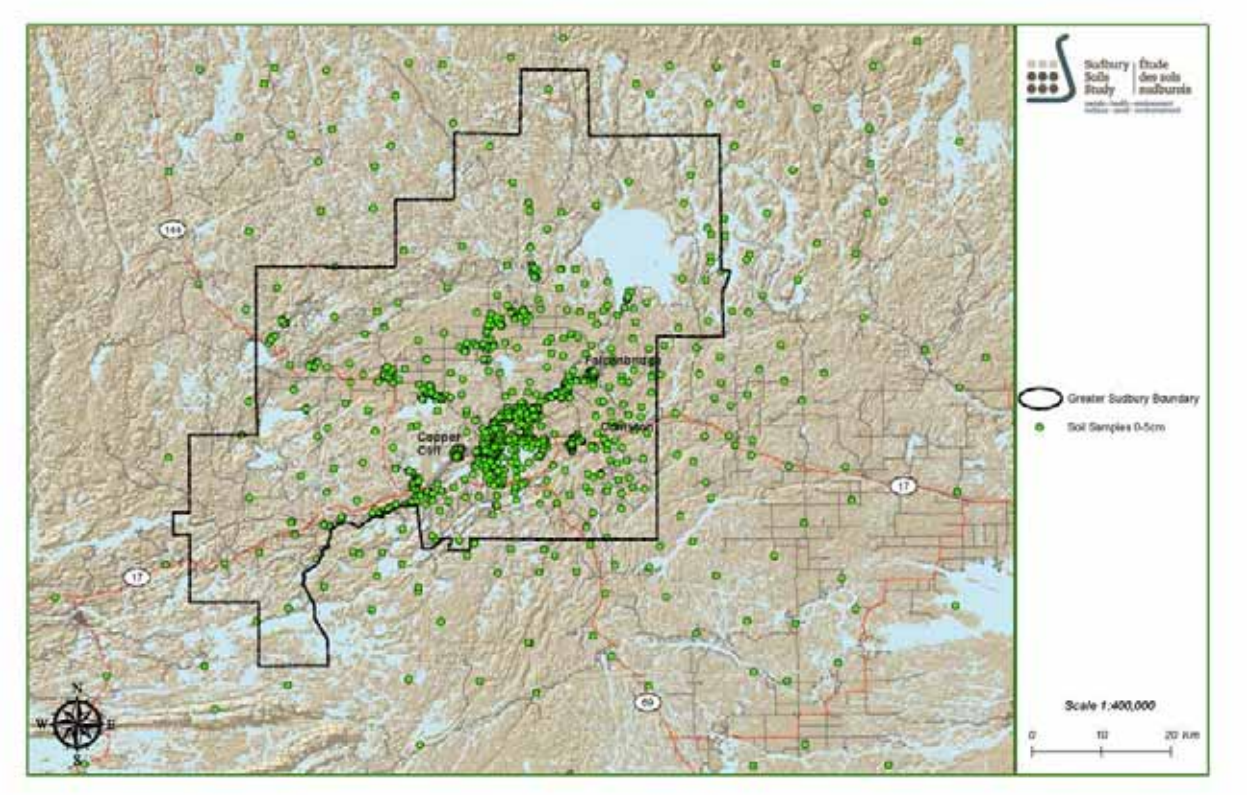


**Figure 4-1 Map of Soil Sampling Locations for Phase I of the Study**

It is important to note that not all of the residential soils samples in the soils database were relevant to the HHRA. Soil samples collected outside of the COI or from non-residential locations were not considered in the soil concentration calculations. Samples taken from the surficial layer of soil at each sampling location (*i.e.*, 0 to 5 cm, 0 to 10 cm, or 0 to 15 cm, depending on the survey methodology) were included in the screening as these top layers of soils are most available for human exposure. Of these samples, a large number were replicate samples and, therefore, were used to calculate a geometric mean for each specific sample location. All soil types were included in the screening with the exception of gravel samples, which were not relevant to the exposure pathways under assessment. Commercial garden and wild soils (*i.e.*, soil collected from areas outside of the urban and suburban areas of the GSA, during the wild blueberry and mushroom sampling program) were also excluded from the screening data set as they are typically amended with various fertilizers and nutrients in the case of commercial gardens, or do not accurately reflect soils with which typical residents of the GSA may come into contact on a daily or frequent basis.

***Data Evaluation for Potential Outliers***

As part of the screening process, the data were also evaluated for the presence of potential statistical outliers which would not be representative of the overall data set. To determine whether a particular sample may be an outlier with respect to the overall data set, each specific "candidate" point was examined using SPSS (version 11) and a box and whisker plot was created to demonstrate its position in relation to the remainder of the data set. In a box and whisker plot, the vertical lines or "whiskers" extending vertically from the bottom and top edges or "hinges" of the box extend out to the minimum and maximum values of the dataset which lie within 1.5-times the interquartile range (IQR) of the hinges of the box (*i.e.*, 1.5 x IQR). The IQR is the difference between the first and third quartile (*i.e.*, Q1 and Q3, respectively). In terms of percentiles, Q1 represents the $25^{th}$ percentile and Q3 represents the $75^{th}$ percentile of a dataset. Statistical convention typically holds that values, which are greater or less than the interquartile range from the top or bottom box hinge (respectively) should be considered as potential outliers (Tukey, 1997). Any points which were determined to be outliers through this method were then excluded from the data set taken forward for further analyses in the HHRA.

This review indicated the presence of three potential outliers within the data set. One replicate soil sample contained an extreme concentration of lead (*i.e.*, 2,600 mg/kg) as compared to the other data points in the 0 to 5 cm layer samples from the Town of Falconbridge (Golder Associates, 2001). A box and whisker plot (Figure 4-2) was created from the dataset composed of all lead concentrations in the 0 to 5 cm layer collected by Golder Associates.

The following values were calculated for the soils dataset:

- Q1 (25th percentile) = 26 mg/kg;
- Q3 (75th percentile) = 70 mg/kg;
- IQR = (Q3-Q1) = 44 mg/kg;
- Lower range of acceptable values = Q1 – 1.5 x IQR = -40 mg/kg (lowest actual value within this limit = 10 mg/kg); and,
- Upper range of acceptable values = Q3 + 1.5 x IQR = 136 mg/kg (highest actual value within this limit = 130 mg/kg).

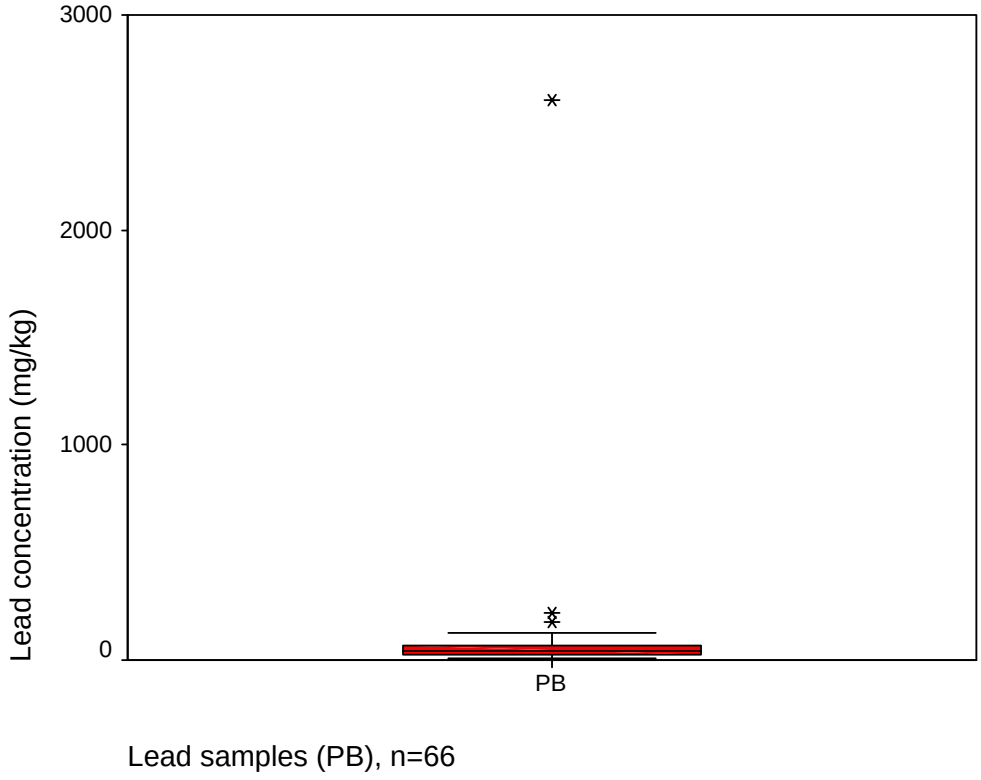


**Figure 4-2 Box plot of lead concentrations in 0 to 5 cm layer samples from Golder Associates sampling in Falconbridge**

The sample in question (2,600 mg/kg) clearly fell outside the IQR. As such, it was decided that this value be classified as an outlier based on visual observation and the foregoing statistical calculations. Based on this analysis, the lead concentration for sample SSG0019 (replicate) of 2,600 mg/kg was removed from the dataset. The lead concentration (*i.e.*, 20 mg/kg) of the original sample (SSG0019 – original) remained as part of the data set and was included in the calculation of the 95% UCLM for lead in the Town of Falconbridge. It should be noted that inclusion of the outlying data point (SSG0019 - replicate) in the calculation of the 95% UCLM would not significantly impact the value of this statistic.

Two other samples in the 0 to 5 cm layer (lead concentration = 180 mg/kg, 220 mg/kg) fell outside the IQR (Figure 4-3). However, these samples were located closer to the Falconbridge stack compared to the other samples (including the excluded lead sample of 2,600 mg/kg) where lead levels were expected to be greater compared to samples further away from the stack. Therefore, these two points were retained as part of the dataset moving forward for further analyses. The whiskers are more evident in Figure 4-3, as is the black line running through the box, which represents the median (40 mg/kg) of the dataset.

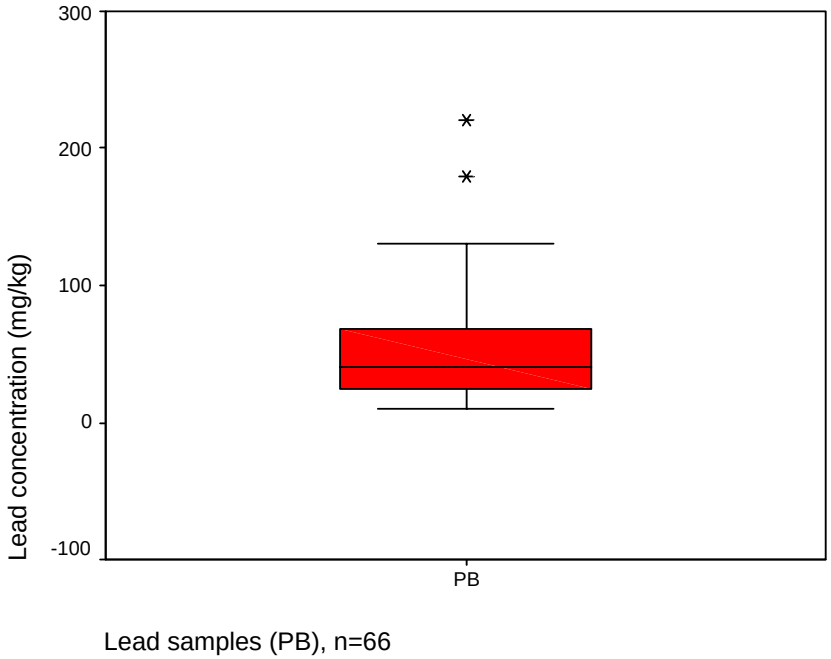


**Figure 4-3 Box plot of lead concentrations in 0 to 5 cm layer samples from Golder Associates Falconbridge sampling with sample containing lead concentration of 2,600 mg/kg removed to show greater detail**

In most soil sampling programs, duplicate samples are collected at one in every 10 sample locations and are split from a thoroughly mixed composite sample made up of two individual samples collected from the site. In those cases, using the arithmetic mean to estimate central tendency between two samples derived from one composite sample is appropriate, as one would be evaluating the systematic or methodological errors (*e.g.*, improper mixing, laboratory errors, *etc*.) which should be normally distributed around the mean. However, this soil database is made up of almost an equal number of original and replicate soil samples from locations across the GSA. In this case, each replicate sample was in fact an additional sample collected from the same location in the same manner as the first. Replicate samples were not homogenized with the original sample to form a composite; rather they represent a second sample collected at the sample site. As not every site had a replicate sample, it was felt that it would unfairly bias the statistics if each of these original and replicate samples were considered individual samples, and not combined to represent that particular sampling location. Therefore, any variance would likely be largely due to minute differences in environmental concentrations at this location. For the purposes of the HHRA, the geometric mean of the original and replicate soil samples was used to calculate COC concentrations in soil. The geometric mean was selected as variation in environmental concentrations would be expected typically to follow a lognormal distributional pattern.

Summary statistics are presented in the Table 4.1 for the soil samples extracted from the database where each sample (original and replicates) is considered as an individual data point. This included the resulting statistics if one treated each of the original and replicate samples as discrete samples (*Complete Data Set*), as well as those related to the arithmetic mean of the original and replicate samples, and the geometric mean of the original and replicate samples. The relative difference between the arithmetic mean and the geometric mean concentration for each COC is also provided, followed by the overall relative difference for all of the COC in the HHRA. A discussion of the uncertainty and implications related to the use of a geometric mean *versus* an arithmetic mean to consolidate sampling data for a specific site is provide in Chapter 7 of this volume.

**Table 4.1 Statistical Summary of Surface Soil COC Concentrations (in mg/kg)**

| COC | No. of Samples (n) | Mean | Standard Deviation | 95% UCLM |
|---|---|---|---|---|
| **ARSENIC** | | | | |
| Complete Data Set | 2137 | 17.3 | 33.1 | 21.8 |
| Arithmetic Mean | 1124 | 17.0 | 33.2 | 23.2 |
| Geometric Mean | 1124 | 16.7 | 32.9 | 22.9 |
| Relative Difference between Means | - | 0.64% | - | 0.60% |
| **COBALT** | | | | |
| Complete Data Set | 2137 | 19.8 | 21.6 | 21.8 |
| Arithmetic Mean | 1124 | 19.3 | 21.1 | 22.0 |
| Geometric Mean | 1124 | 19.2 | 21.0 | 21.9 |
| Relative Difference between Means | - | 0.27% | - | 0.27% |
| **COPPER** | | | | |
| Complete Data Set | 2137 | 427.3 | 669.8 | 517.8 |
| Arithmetic Mean | 1124 | 411.0 | 654.4 | 532.9 |
| Geometric Mean | 1124 | 407.4 | 651.6 | 528.7 |
| Relative Difference between Means | - | 0.44% | - | 0.39% |
| **LEAD** | | | | |
| Complete Data Set | 2136 | 45.8 | 56.9 | 53.5 |
| Arithmetic Mean | 1124 | 44.4 | 55.5 | 54.7 |
| Geometric Mean | 1124 | 43.8 | 55.0 | 54.1 |
| Relative Difference between Means | - | 0.60% | - | 0.57% |
| **NICKEL** | | | | |
| Complete Data Set | 2137 | 398.1 | 543.2 | 471.5 |
| Arithmetic Mean | 1124 | 383.7 | 531.2 | 482.6 |
| Geometric Mean | 1124 | 380.3 | 528.2 | 478.7 |
| Relative Difference between Means | - | 0.43% | - | 0.40% |
| **SELENIUM** | | | | |
| Complete Data Set | 2137 | 2.25 | 3.58 | 2.74 |
| Arithmetic Mean | 1124 | 2.18 | 3.46 | 2.82 |
| Geometric Mean | 1124 | 2.14 | 3.43 | 2.78 |
| Relative Difference between Means | - | 0.89% | - | 0.78% |
| **OVERALL RELATIVE DIFFERENCE** | **-** | **0.55%** | **-** | **0.50%** |

In addition to the soil surveys, several other surveys were conducted to collect Sudbury-specific media concentration data for the purpose of filling data gaps and for use in the ERA and HHRA (see study overviews outlined in Chapter 3 and detailed reports provided in the appendices to this volume). For the

HHRA, residential soil samples collected during the Vegetable Garden Survey (Appendix E) and the Indoor Dust Survey (Appendix M) were incorporated into the soil concentration data set for calculation of a 95% UCLM for COC concentrations in each COI. A total of 55 additional soil samples were added from the Vegetable Garden Survey, and 86 from the Indoor Dust Survey, to the overall assessment data set. As before, the geometric mean soil concentration was calculated and utilized for replicate soil samples, where applicable.

Analysis of the complete soils datasets for each COI in the HHRA indicated a notable proportion of arsenic and selenium levels detected at, or below, the minimum detection limits (MDLs) of 5.0 µg/g and 1.0 µg/g, respectively. Of the total 1,124 soil samples reviewed for the HHRA from the soils database, 35% (389 samples) of the arsenic concentrations and 49.5% (553 samples) of the selenium concentrations were at, or below, the respective MDLs (geometric means calculated for original and replicate samples where both samples were <MDL). Soil sample analysis for the Vegetable Garden Survey (Appendix E) employed lower MDLs than those in the original soil surveys, resulting in no arsenic concentrations at less than the MDL (0.5 µg/g) and only seven samples with selenium concentrations below the MDL (0.5 µg/g). The results of the Indoor Dust Survey (Appendix M) indicated approximately 38% of samples below the MDL for selenium (0.8 µg/g) in soil. None of the dust survey soil samples contained levels of arsenic below the MDL of 0.6 µg/g for arsenic.

The following table provides a summary of the number of soil samples with non-detect concentrations of arsenic and selenium in each COI.

**Table 4.2 Statistical Summary of Soil COC Concentrations for Arsenic and Selenium below the Analytical MDL**

| COI | Total No. of Samples in Data Set | ARSENIC | | SELENIUM | |
|---|---|---|---|---|---|
| | | No. of Samples <MDL | Percentage of Total Data Set | No. of Samples <MDL | Percentage of Total Data Set |
| **MDLs (µg/g)** | | ***5.0 [a], 0.5 [b], 0.6 [c]*** | | ***1.0 [a],0.5 [b], 0.8 [c]*** | |
| Coniston | 203 | 44 | 21.2% | 111 | 54.7% |
| Copper Cliff | 197 | 12 | 6.1% | 8 | 4.1% |
| Falconbridge | 188 | 10 | 5.3% | 25 | 13.3% |
| Sudbury Centre | 597 | 273 | 45.7% | 378 | 63.3% |
| Hanmer | 80 | 50 | 62.5% | 72 | 90.0% |

[a] MDL used in analysis of samples from Sudbury Soils Study (SARA, 2004).
[b] MDL used in analysis of samples from Vegetable Garden Survey (Appendix E).
[c] MDL used in analysis of samples from Indoor Dust Survey (Appendix M).

In general, the arithmetic mean or average is the preferred statistic used to describe the central location in most data sets. The advantages of using the arithmetic mean are that it is easy to calculate, has a smaller standard error than other statistics of location, and tends to be normally distributed when the original data are not (Sokal and Rohlf, 1981). The arithmetic mean, however, is sensitive to changes in the shape of a frequency distribution and can be shifted in either direction by the presence of outliers or a large number of data points with equal values (*e.g.*, non-detect values). In cases when a data set is skewed, the most meaningful descriptor of central location is the median value. The median value divides a frequency distribution into two equal halves and is most often used to describe data sets that are not normally distributed (Sokal and Rohlf, 1981).

The greater part of the soils datasets for each COC in each of the COI are not normally distributed. As shown in Table 4.2, some datasets are skewed due to the large proportion of values recorded at or below the detection limit, while other datasets are impacted by the presence of outliers.

In order to compare and contrast the COC soil levels among the five COI, the following box plots were created using the complete soil datasets from each community. The plots were created using XlstatPro software which indicates central tendency, variability, symmetry of distribution and the presence of outliers in data sets. The following box plot diagrams (Figures 4-4 to 4-8) include:

- The average (arithmetic mean) soil concentration which is identified as the red line and is provided adjacent to the box;
- The median soil concentration which is identified as the black line and is provided adjacent to the box;
- The first (Q1 = $25^{th}$ percentile) and third (Q3 = $75^{th}$ percentile) quartiles of the data set which are indicated by the top and bottom of the boxes; and,
- The lower bound ($I_{Q1}$) and upper ($I_{Q3}$) bounds of the IQR which are represented by the upper and lower whiskers, or cross hairs, and are calculated using the following equations:

$$I_{Q1} = Q1 - [1.5 \times (Q3 - Q1)]$$
$$I_{Q3} = Q3 + [1.5 \times (Q3 - Q1)]$$

- Soil concentrations outside of the first and third quartiles are represented by individual circles. The circles that are filled in (black dots) represent soil concentrations that are more than three times the IQR. Empty circles represent concentrations within this range.

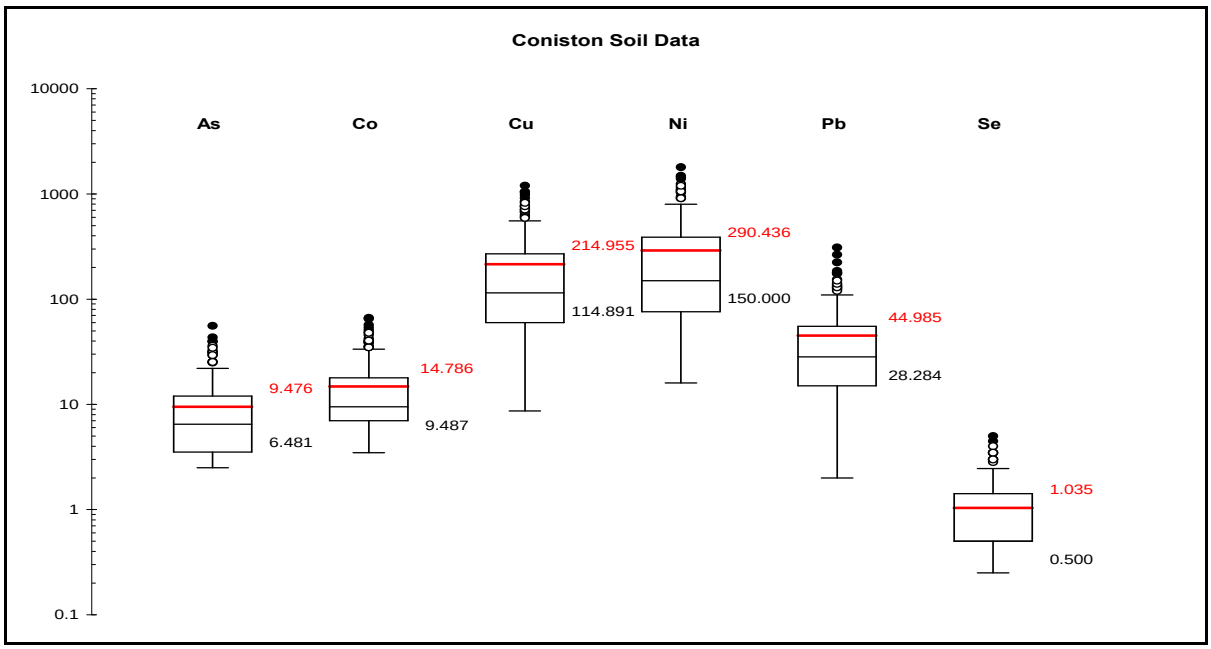


**Figure 4-4 Box plot diagram of COC soil concentrations in Coniston**

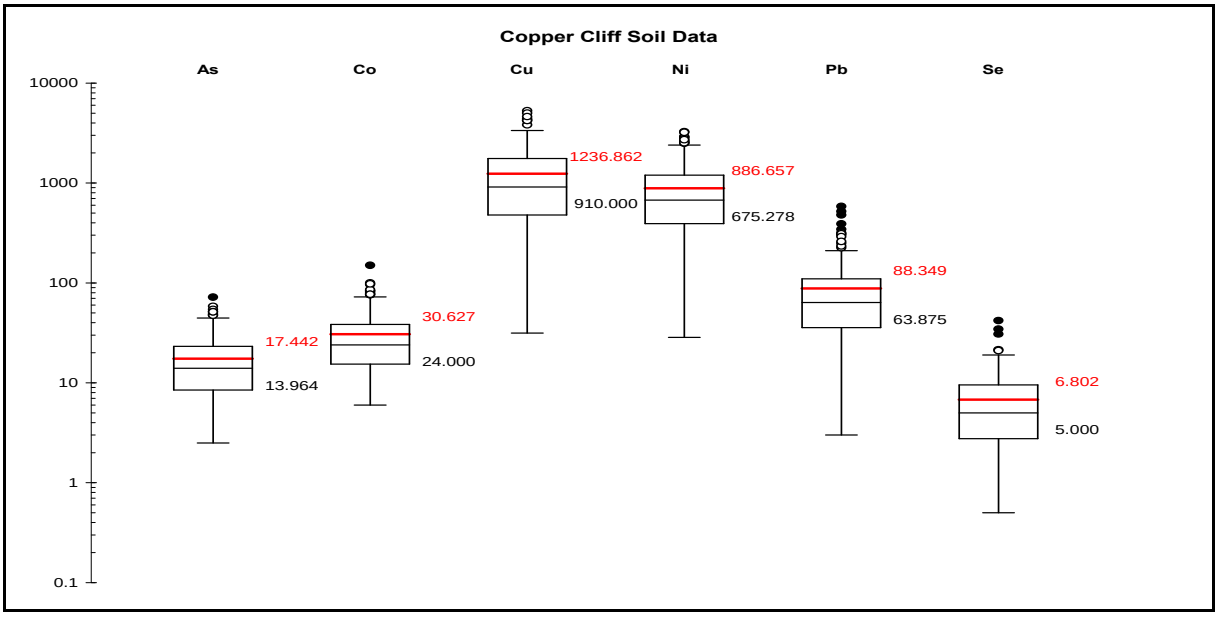


**Figure 4-5 Box plot diagram of COC soil concentrations in Copper Cliff**

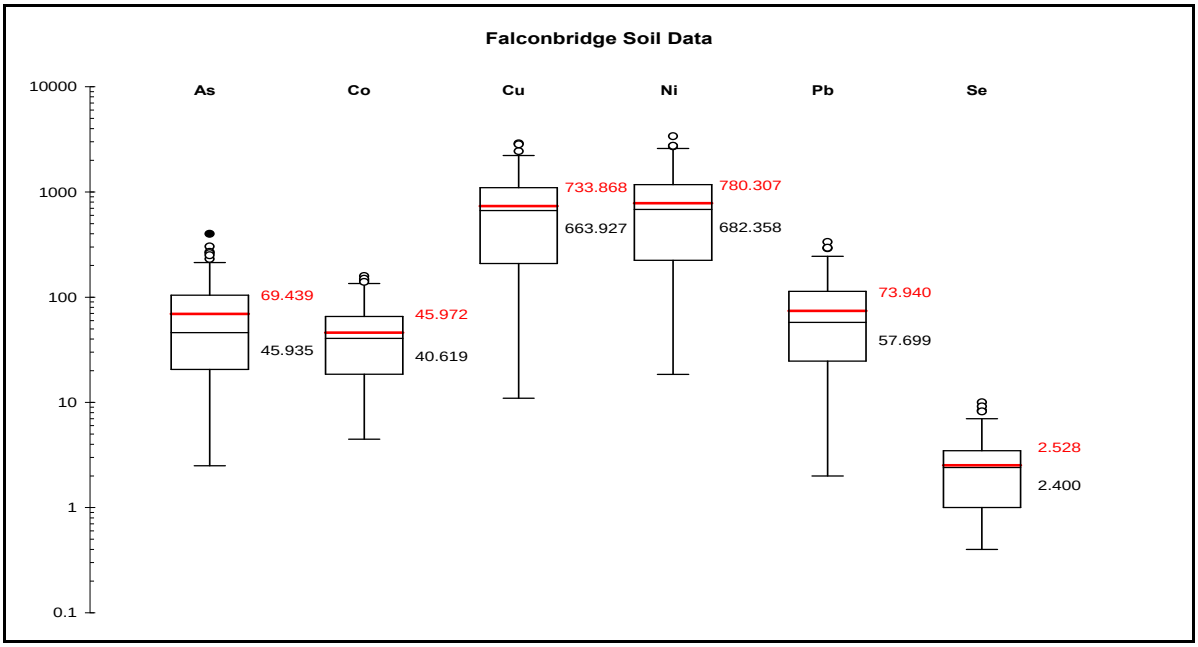


**Figure 4-6 Box plot diagram of COC soil concentrations in Falconbridge**

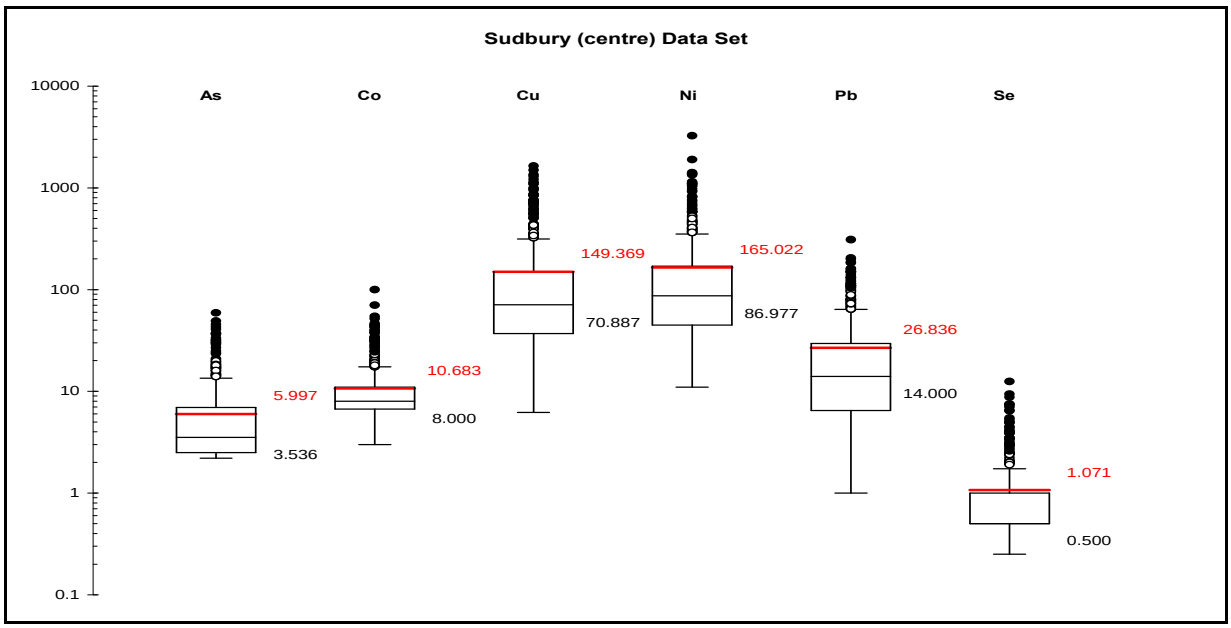


**Figure 4-7 Box plot diagram of COC soil concentrations in Sudbury centre**

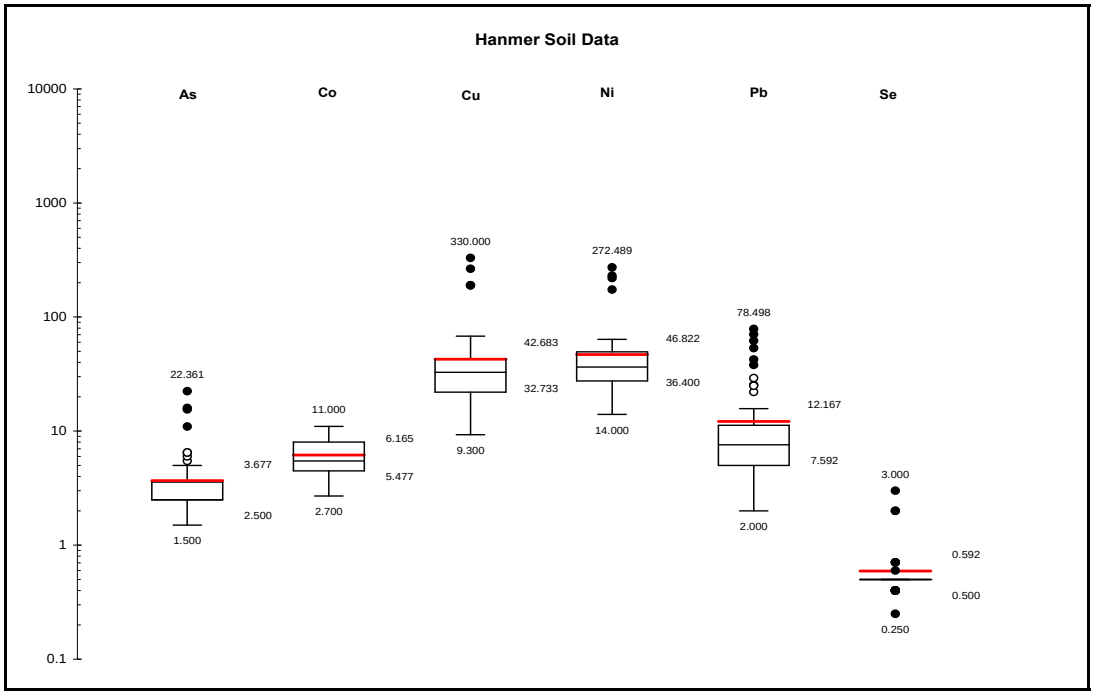


**Figure 4-8 Box plot diagram of COC soil concentrations in Hanmer [a]**

[a] The missing box for selenium concentrations is due to the more than 90% of samples measured at less than the detection limit, which made it difficult to calculate Q1 and Q3.

The box plots provide a good picture of the soil data sets for each community and allow for comparison of soil COC levels between COI. A few observations should be noted following review of the data. The levels of arsenic in each community appear to be within a similar range, with Falconbridge having higher concentrations and Hanmer and Sudbury Centre on the lower end of the range, both having a large percentage of values below the detection limit. Cobalt concentrations in soil seem to be uniformly distributed in each COI, with higher concentrations measured in Falconbridge and lower levels measured in Sudbury Centre and Hanmer. Soil copper levels appear to be skewed towards the higher concentrations, due to a greater proportion of samples in the higher concentration range. A greater number of samples with elevated concentrations of copper also results in a median value that is close to the 75$^{th}$ percentile (Q3). Nickel concentrations in soil vary greatly across the GSA and within each data set. The datasets for nickel tend to be skewed towards the higher concentrations, due to the large proportion of elevated soil samples. Lead levels appear to be within a similar range among the COI, with slightly higher levels in Copper Cliff and Falconbridge. Finally, the levels of selenium in soil are difficult to depict due to a high proportion of samples detected below the detection limit. The box missing for

selenium concentrations for Hanmer is due to the more than 90% of samples measured at less than the detection limit, thereby making it difficult to calculate Q1 and Q3.

In general, the box plots indicate that soil levels in each of the COI with smelters (historically or presently) tend to have higher concentrations than those communities without smelter operations in direct proximity, such as is the case with Sudbury Centre and Hanmer. The plot for the Sudbury Centre community indicates a number of values greater than the $I_{Q3}$ value. This is due to a much larger dataset for Sudbury centre than for the other COI in which the vast majority of samples fall within a small range. The IQR is quite small which results in $I_{Q1}$ and $I_{Q3}$ values that are not very far reaching. A more detailed analysis of the soils data is provided in Volume 1 of the Sudbury Soils Study report.

Table 4.3 provides a summary of the COC concentrations in surface soils for each COI in the HHRA.

**Table 4.3 Summary of Surface Soil Concentrations in the GSA (µg/g)**

| COI | COC | Min | Max | Mean (arithmetic) | 95% UCLM | 95th Percentile |
|---|---|---|---|---|---|---|
| **Coniston** (n=203) | As | 2.50 | 55.7 | 9.48 | 12.2 | 29.3 |
| | Co | 3.46 | 66.8 | 14.8 | 18.5 | 40.2 |
| | Cu | 8.64 | 1200 | 215 | 315.5 | 709.9 |
| | Ni | 16.0 | 1800 | 290.4 | 432.8 | 1043 |
| | Pb | 2.00 | 309.8 | 45.0 | 52.0 | 139.2 |
| | Se | 0.25 | 5.00 | 1.04 | 1.31 | 3.00 |
| **Copper Cliff** (n=197) | As | 2.50 | 72.0 | 17.4 | 19.0 | 41.5 |
| | Co | 6.00 | 150.0 | 30.6 | 33.4 | 77.3 |
| | Cu | 31.4 | 5290 | 1240 | 1370 | 3042 |
| | Ni | 28.5 | 3260 | 886.7 | 976.1 | 2505 |
| | Pb | 3.00 | 582.4 | 88.4 | 97.9 | 251.4 |
| | Se | 0.50 | 42.0 | 6.80 | 7.51 | 16.9 |
| **Falconbridge** (n=188) | As | 2.50 | 400.0 | 69.4 | 78.7 | 204.8 |
| | Co | 4.47 | 159.0 | 46.0 | 56.5 | 106.4 |
| | Cu | 11.0 | 2900 | 733.9 | 1010 | 1774 |
| | Ni | 18.4 | 3390 | 780.3 | 1070 | 1990 |
| | Pb | 2.00 | 335 | 73.9 | 82.3 | 191.4 |
| | Se | 0.40 | 10.0 | 2.53 | 3.09 | 5.76 |
| **Sudbury Centre** (n=597) | As | 2.20 | 59.0 | 6.00 | 7.17 | 17.4 |
| | Co | 3.00 | 100.0 | 10.7 | 11.3 | 25.4 |
| | Cu | 6.20 | 1640 | 149.4 | 204.0 | 569.2 |
| | Ni | 11.0 | 3260 | 165.0 | 210.1 | 578.7 |
| | Pb | 1.00 | 309.8 | 26.8 | 35.9 | 109.4 |
| | Se | 0.25 | 12.5 | 1.07 | 1.30 | 3.46 |
| **Hanmer** (n=80) | As | 1.50 | 22.4 | 3.68 | 4.27 | 6.70 |
| | Co | 2.70 | 11.0 | 6.16 | 6.55 | 10.0 |
| | Cu | 9.30 | 330.0 | 42.7 | 67.0 | 74.1 |
| | Ni | 14.0 | 272.5 | 46.8 | 67.9 | 69.2 |
| | Pb | 2.00 | 78.5 | 12.2 | 19.2 | 43.1 |
| | Se | 0.25 | 3.00 | 0.59 | 0.68 | 0.77 |

n = Number of samples analysed.

As can be noted in Figures 4-4 through 4-8, and by the differences between the 95 UCLM (representing average community concentrations) and the 95th percentile (representing worst-case property-specific concentrations) of the distribution of soil concentrations within each COI, some properties may have higher concentrations than those found on average throughout the entire COI. The evaluation of risks related to exposures to both average community soil concentrations, and maximum soil concentrations for lead are discussed in Chapter 5.

#### 4.1.1.2 Ambient Air Concentrations

RWDI, as part of the SARA Group, completed a year-long air monitoring program to collect Sudbury-specific ambient air concentrations for all seasons under variable wind and climate conditions. A brief summary of the air monitoring program was presented in Chapter 3 (Section 3.3) of this report. The complete City of Greater Sudbury Air Monitoring Network Report is located in Appendix F.

Air monitoring was conducted at nine sites in the GSA and at one "background" location (Windy Lake Provincial Park) just outside the boundary of the GSA. Air data were collected from populated areas in each of the COI including two locations in Sudbury Centre (at the west and south end). High and low volume samplers were used to collect three size fractions of particulate matter on quartz fibre filters: total suspended particulate matter (TSP), particulate matter less than 10 microns ($PM_{10}$), and particulate matter less than 2.5 microns ($PM_{2.5}$). These size fractions were selected for sampling because they are most relevant to human exposure and can be retained in the nose (TSP), upper lung ($PM_{10,}$) and lower lung ($PM_{2.5}$) of an individual. While the $PM_{2.5}$ particulate fraction has the potential to penetrate deepest into the lungs, and much of the recent regulatory interest is being focussed on this particular fraction for the evaluation of health impacts, for the current assessment $PM_{10}$ concentrations were used to calculate 95% UCLM values for each COC because this fraction conservatively represents the most toxicologically significant particle size for human exposure and toxicity (*i.e.*, both the $PM_{2.5}$ fraction and slightly larger particles which may cause impacts/irritation within the upper lungs).

Table 3.5 in Chapter 3 of this Volume provides summary statistics and 95% UCLM values for each COI for ambient air concentrations used in the current assessment.

#### 4.1.1.3 Indoor Air Concentrations

Indoor air concentrations were assumed to be equal to measured outdoor air concentrations. This is thought to be a conservative assumption as a number of recent studies (Chao and Wong, 2002; Komarnicki, 2005; Molnar *et al*., 2005) demonstrate that outdoor concentrations of heavy metals can be significantly greater than measured indoor air concentrations. Lower indoor air concentrations appear to be a result of outdoor air filtration as the outdoor air infiltrates indoor environments and dilution with the existing indoor aerosol. Although there do appear to be some minor indoor sources of metals, their contribution does not appear to be significant compared to the contribution of outdoor air.

#### 4.1.1.4 Drinking Water Concentrations

Potable water in the GSA originates from both groundwater and surface water sources and is provided by municipal and private systems. The majority of homes in the GSA (88%) are serviced by municipal drinking water in each of the five COI. For the current assessment, drinking water monitoring data were collected and reviewed from a number of sources for use in the estimate of an individual's exposure to COC through the consumption of drinking water. The following section describes the drinking water datasets reviewed in the current assessment.

***Provincial Water Monitoring Data***

To estimate human exposure to COC in drinking water, monitoring data were obtained through the Drinking Water Surveillance Program (DWSP), a voluntary monitoring program managed by the MOE in conjunction with municipalities across Ontario. Unfiltered, total metal concentration data collected from 1995 through 2005 from water supply systems and water treatment plants that service each COI were used to calculate a 95% UCLM for human exposure to each COC through consumption of residential drinking water. A ten-year span of water data was selected to ensure sufficient quantity of data representing seasonal and yearly cycling, yet recent enough to properly represent a reasonable reflection of current and near-future water quality. Figure 4-9 provides a map of the various municipal water pipeline systems servicing the GSA.

Water samples collected from distribution systems (free flowing) were included in the exposure estimates. The DWSP dataset typically includes concentration data from three specific locations within the distribution system: 1) raw water (prior to entering the water treatment plant); 2) treated (after exiting the water treatment plant); and, 3) distribution (after it has entered the municipal potable water distribution system). Since this water is treated and circulated through the distribution pipelines, the distribution data points most accurately represent residential tap water consumed by individuals (*i.e.,* from a kitchen faucet). Therefore, data on raw and treated water samples within the DWSP database were excluded from the exposure estimates for drinking water as they are more representative of water quality before treatment has occurred (*i.e.*, raw) and prior to circulation through the water supply system (*i.e.*, raw and treated).

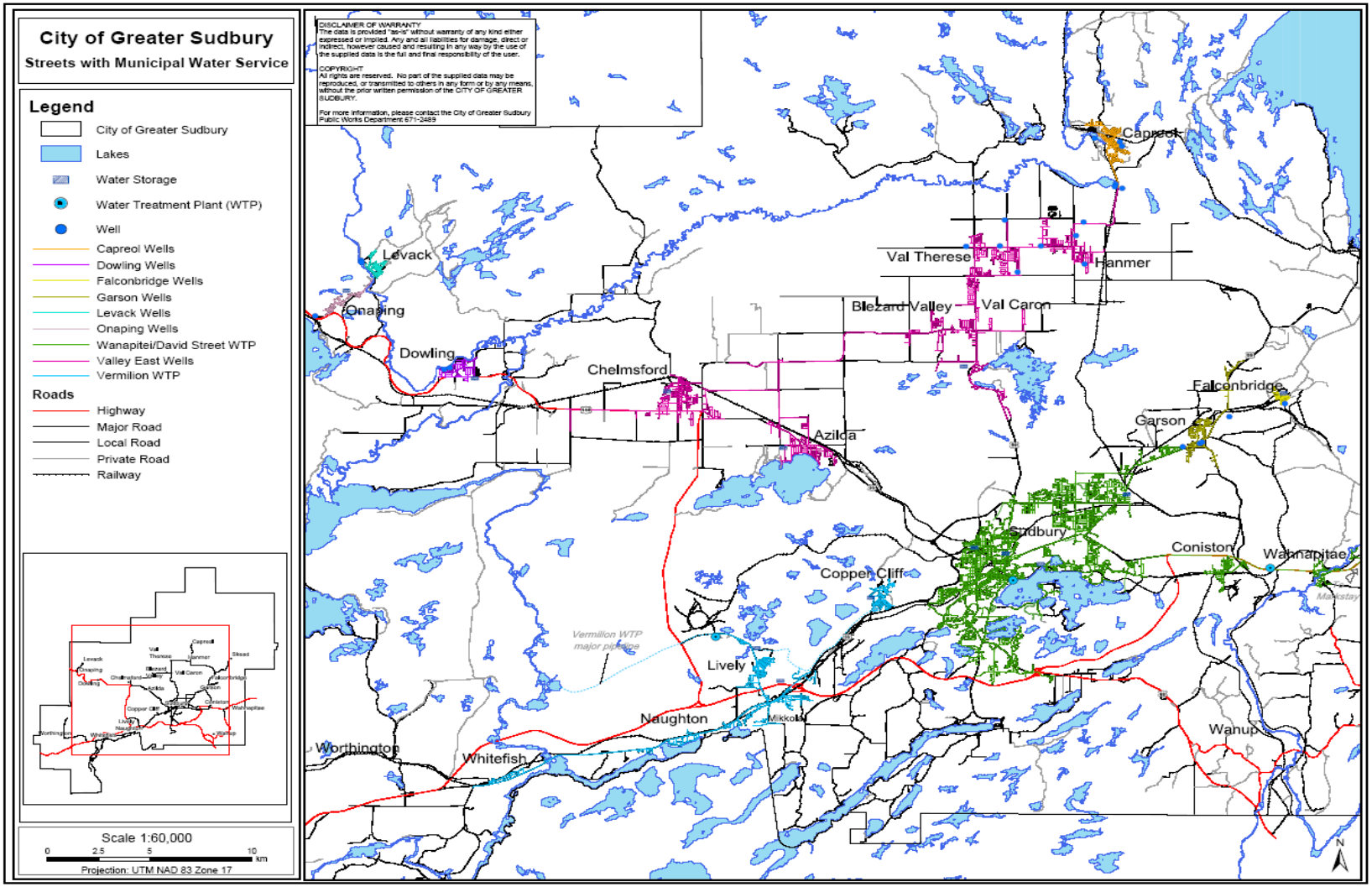


**Figure 4-9 City of Greater Sudbury Streets with Municipal Water Services (CGS, 2004a, pers. comm.)**

Review of the DWSP data sets indicated several negative and zero concentration values. Correspondence with the MOE (Fellows, 2005 pers. comm.) indicated that negative concentration values were due to corrections made to the data set based on laboratory blanks or interference and do not represent any errors in the data set. For the purpose of statistical analyses of the data set, any negative concentration or zero value was set equal to half the detection limit. Applying half the detection limit to the negative concentration and zero values is considered to be a conservative method to evaluate the data set provided.

Drinking water is supplied to residents of Coniston *via* the Wanapitei water treatment plant (WTP), which is owned and operated by the City of Greater Sudbury and draws water from the Wanapitei River. Potable water for the residents of Sudbury centre is provided by the municipality of the City of Greater Sudbury *via* both the Wanapitei WTP and the David Street WTP, which draws water from Lake Ramsey. Unfiltered, total metal concentrations in potable water collected between 1995 and 2005 from distribution systems of both Wanapitei and David Street WTPs were provided by the MOE from data collected as part of DWSP (McMahon, 2005 pers. comm.). These data were used to estimate 95% UCLM values for drinking water for both of these COI.

Data for unfiltered total metal concentrations from Blezard Valley Well Supply between 1995 and 2005 were used in the calculation of a 95% UCLM for each COC in drinking water in Hanmer. The Blezard Valley Well Supply is owned and operated by the City of Greater Sudbury and uses groundwater as a source of potable water for several communities including Hanmer. These data were provided to the SARA Group by the MOE from data collected as part of the DWSP (McMahon, 2005 pers. comm.).

The drinking water supply system servicing the Town of Falconbridge is owned and operated by Xstrata Nickel and is not part of the DWSP. Ontario Regulation 170/03 requires owners and operators of drinking water systems to ensure that the water meets prescribed drinking water standards along the entire system. Analytical test results for drinking water operations are reported to the MOE for specific parameters listed in the regulation, including arsenic, lead and selenium. These analytical results then become part of the MOE's Drinking Water Information System (DWIS) and Drinking Water Database (DWWS). The regulation does not require monitoring for cobalt or nickel. For the current assessment, unfiltered, total metal concentrations in drinking water from the Falconbridge Well Supply and Distribution System recorded in the DWIS database were provided to the SARA Group for periodic monitoring conducted between 2002 and 2004 for arsenic, copper, lead, and selenium. In addition to the DWIS data, Xstrata Nickel provided water monitoring data collected from 1995 through 2005 for total metal concentrations of copper, nickel and arsenic at Deep Well No. 4. The water concentration data

from DWIS and from Xstrata Nickel were combined to estimate a 95% UCLM for drinking water consumed by residents of the Town of Falconbridge.

In the summer of 2005, the drinking water source supplying the Town of Falconbridge was switched from the original well to a new deeper well. Initial sampling data, including MOE audits, indicate that concentrations of lead have greatly decreased with the change to the new well. While both sources are displayed in Table 4.4 below, as the intention is to evaluate current risks for the residents of Falconbridge and in the near future, exposures to drinking water from the new well were used in the current assessment.

Neither the DWIS dataset nor the monitoring data provided by Xstrata Nickel included concentration data for levels of cobalt in drinking water. Since Ont. Reg. 170/03 does not require cobalt to be monitored in potable water, concentration data for this element in drinking water in Falconbridge do not exist.

To characterize exposure to cobalt in potable water for residents of the Town of Falconbridge, cobalt concentrations measured in drinking water for Sudbury Centre and Coniston (same dataset) were used as a surrogate to estimate a 95% UCLM value for cobalt in drinking water in Falconbridge. Sudbury Centre and Coniston represent the closest COI for which cobalt data in drinking water are available and, therefore, were considered the most appropriate surrogate dataset for this evaluation.

The community of Copper Cliff is serviced by drinking water from the Vermilion River Water Treatment Plant and Vermilion Distribution System (DS). The WTP is owned and operated by Vale Inco and is not part of the DWSP. For the current assessment, drinking water monitoring data for the Vermilion WTP and DS were provided by the MOE from the DWIS database for unfiltered concentrations of arsenic, copper, lead, and selenium, for 2001 through 2004. In addition, Vale Inco provided results of more recent analyses of distributed drinking water collected from the Copper Cliff area. The DWIS data set and the Vale Inco data set were combined to estimate a 95% UCLM for drinking water concentrations of each COC that would be consumed by residents of the community of Copper Cliff.

Table 4.4 provides a summary of COC concentrations in potable water used in current assessment.

**Table 4.4 Summary of Drinking Water Concentrations in the GSA (µg/L)**

| COI | COC (No. of Samples) | | Min | Max | Mean[a] | Standard Deviation | 95% UCLM | 95th Percentile |
|---|---|---|---|---|---|---|---|---|
| **Coniston** | As (62) | | 0.20 | 2.00 | 0.87 | 0.50 | 1.14 | 1.7 |
| | Co (62) | | 0.01 | 2.20 | 0.16 | 0.29 | 0.20 | 0.44 |
| | Cu (62) | | 1.50 | 212 | 36.2 | 37.2 | 44.6 | 96.2 |
| | Ni (62) | | 6.80 | 120 | 35.3 | 31.7 | 52.8 | 97.2 |
| | Pb (62) | | 0.03 | 1.67 | 0.26 | 0.26 | 0.31 | 0.73 |
| | Se (62) | | 0.31 | 5.00 | 0.91 | 0.67 | 1.28 | 2 |
| **Copper Cliff** | As (9) | | 0.25 | 4.50 | 1.23 | 1.37 | 2.53 | 3.5 |
| | Co (4) | | 0.02 | 0.05 | 0.03 | 0.011 | 0.05 | 0.045 |
| | Cu (7) | | 18.3 | 248 | 103 | 90.8 | 170 | 226 |
| | Ni (4) | | 8.37 | 49.3 | 20.1 | 19.6 | 49.3 | 43.9 |
| | Pb (9) | | 0.30 | 2.80 | 0.82 | 0.79 | 1.39 | 2.08 |
| | Se (9) | | 0.50 | 3.00 | 1.66 | 1.12 | 3.00 | 2.8 |
| **Falconbridge** | As (194) | | 0.40 | 5.70 | 2.46 | 0.90 | 2.57 | 4 |
| | Co[b] (62) | | 0.01 | 2.20 | 0.16 | 0.29 | 0.20 | 0.44 |
| | Cu (421) | | 1.00 | 500 | 18.9 | 37.7 | 30.4 | 100 |
| | Ni (431) | | 2.00 | 160 | 30.4 | 16.4 | 31.7 | 60 |
| | Pb | Old well (10) | 0.60 | 6.00 | 1.46 | 1.60 | 3.67 | 3.75 |
| | | New well (7) | 0.18 | 1.4 | 0.50 | 0.44 | 0.97 | 1.19 |
| | Se (3) | | 2.50 | 2.50 | NA | 0 | 2.50 | NA |
| **Sudbury Centre** | As (62) | | 0.20 | 2.00 | 0.87 | 0.50 | 1.14 | 1.7 |
| | Co (62) | | 0.01 | 2.20 | 0.16 | 0.29 | 0.20 | 0.44 |
| | Cu (62) | | 1.50 | 212 | 36.2 | 37.2 | 44.6 | 96.2 |
| | Ni (62) | | 6.80 | 120 | 35.3 | 31.8 | 52.8 | 97.2 |
| | Pb (62) | | 0.03 | 1.67 | 0.26 | 0.26 | 0.31 | 0.73 |
| | Se (62) | | 0.31 | 5.00 | 0.91 | 0.67 | 1.28 | 2 |
| **Hanmer/ Val Therese** | As (18) | | 0.90 | 1.96 | 1.33 | 0.301 | 1.46 | 1.83 |
| | Co (18) | | 0.01 | 0.14 | 0.04 | 0.031 | 0.06 | 0.081 |
| | Cu (18) | | 0.50 | 115 | 53.1 | 29.4 | 65.2 | 96.0 |
| | Ni (18) | | 0.10 | 2.10 | 0.29 | 0.47 | 0.80 | 0.66 |
| | Pb (18) | | 0.05 | 1.14 | 0.34 | 0.31 | 0.49 | 1.05 |
| | Se (18) | | 0.12 | 2.00 | 0.87 | 0.40 | 1.28 | 1.15 |

[a] Arithmetic mean
[b] Cobalt concentrations from Wanapitei and David Street WTPs used as surrogate.
NA = not applicable

***Municipal Water Monitoring Data***

Drinking water monitoring data collected by municipally-owned and operated water works in the GSA are reported in *The City of Greater Sudbury 2003 Annual Water Works Report* (CGS, 2004b). Concentration data in the Annual Water Works Report were reviewed for the current assessment; however, the dataset was not included in the calculation of 95% UCLM values for COC in drinking water, as discussed below.

In the Annual Water Works Report (CGS, 2004b) analytical results for four of the six COC in the HHRA are reported for each quarter year (*i.e.,* once every three months). The report does not indicate whether the samples were taken from treated or distributed water sources or what type of samples were analyzed (*i.e.,* filtered *versus* unfiltered). Additionally, the set is incomplete, having concentrations missing for some COC and some quarters and detection limits are not reported at all. Based on reporting and sampling differences between the Annual Water Works report and the DWSP data sets, combining the data would be difficult and inaccurate. Therefore, following the review of the Annual Water Works dataset, the decision was made to exclude it from the drinking water dataset for estimation of human exposure to COC in drinking water.

***Drinking Water Survey of Private Residential Water Supplies***

One area of uncertainty identified in the HHRA was metal levels in private drinking water supplies in the GSA. The majority of households in the GSA (88%) are serviced by the municipal drinking water supply for which monitoring and concentration data are collected on a regular basis (see above). However, metal concentration data for households serviced by private water supplies, either by surface or ground water, were deficient for the GSA. In order to collect site-specific data on the range of COC concentrations found in private residential drinking water in the Sudbury area, the SARA Group conducted a voluntary drinking water survey for households with private water supplies.

Water samples were collected from 76 private groundwater wells and 18 surface water intakes in the Sudbury area. Tap water samples were collected following a two minute flushing period flow and were analysed for total metal concentrations. A brief summary of the Drinking Water Survey Data Report is located in Section 3.5 of Chapter 3 while the full report can be found in Appendix L of this volume.

Table 4.5 provides a summary of the 95% UCLM values for each COC for private water supplies in the GSA.

**Table 4.5 Drinking Water Survey Concentrations (µg/L)**

| Potable Water Source | COC | Min | Max | Mean[a] | Standard Deviation | 95% UCLM | 95th Percentile |
|---|---|---|---|---|---|---|---|
| **Groundwater** (n=76) | As | 1.00 | 23.0 | 2.37 | 4.0 | 3.27 | 9.0 |
| | Co | 0.15 | 8.70 | 0.56 | 1.4 | 0.88 | 2.75 |
| | Cu | 0.25 | 216 | 45.1 | 53.7 | 57.4 | 148 |
| | Ni | 0.50 | 123 | 11.2 | 27.3 | 17.4 | 70.3 |
| | Pb | 0.05 | 8.00 | 0.70 | 1.3 | 0.99 | 2.08 |
| | Se[b] | 1.50 | 1.50 | 1.50 | 0 | NA | 1.50 |
| **Lake water** (n=18) | As[b] | 1.00 | 1.00 | 1.00 | 0 | NA | 1.00 |
| | Co | 0.15 | 0.40 | 0.16 | 0.059 | 0.19 | 0.19 |
| | Cu | 20.9 | 302 | 97.7 | 70.6 | 133 | 215 |
| | Ni | 9.96 | 126 | 56.8 | 22.5 | 67.9 | 85.4 |
| | Pb | 0.20 | 5.00 | 1.46 | 1.28 | 2.09 | 3.47 |
| | Se[b] | 1.50 | 1.50 | 1.50 | 0 | NA | 1.58 |

n - Number of samples analysed.
NA – Not applicable. Statistic could not be calculated with the given data set.
[a] Arithmetic mean
[b] All samples were below the detections limit; arsenic MDL = 2.0 µg/L, selenium MDL = 3.0 µg/L. In these cases, ½ the detection limit was used as the 95% UCLM.

In certain cases, such as with arsenic and selenium concentrations in lake water and with selenium concentrations in groundwater wells, all water samples had concentrations below the respective minimum detection limits (MDL = 2 µg/L for arsenic; MDL = 3 µg/L for selenium). Based on the data for these COC, mean and 95% UCLM values could not be calculated and have been replaced with half the MDL.

For the current assessment, potable water concentrations in private sources collected during the Drinking Water Survey were compared with COC levels in the municipal drinking water systems for each COI. The following figures depict COC levels in well water, lake water and municipally-supplied drinking water in each COI.

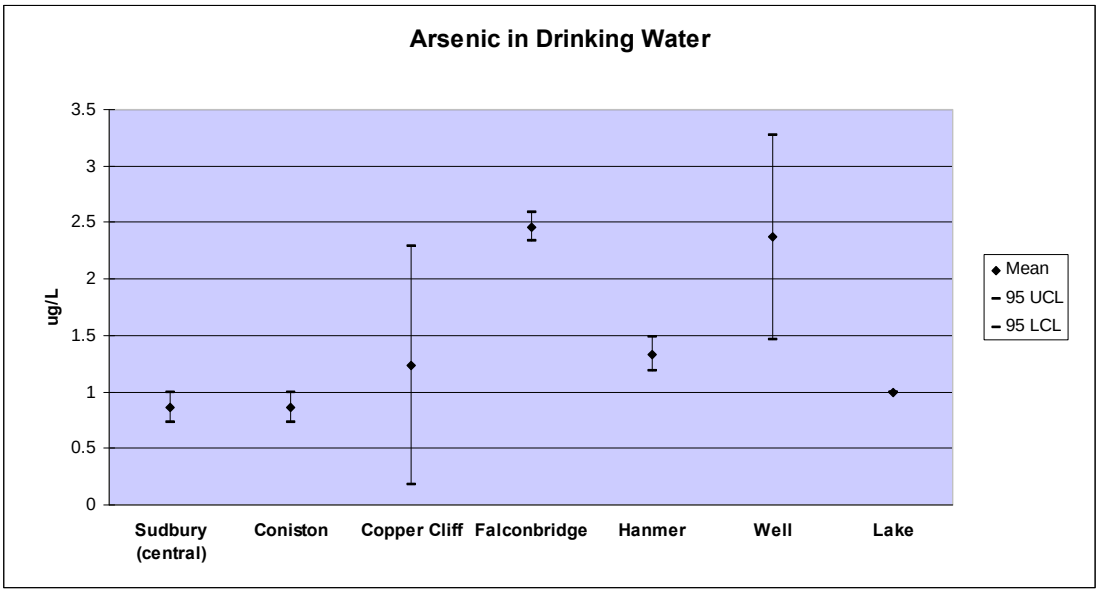


**Figure 4-10 Arsenic Concentrations in Drinking Water in the GSA (µg/L)**

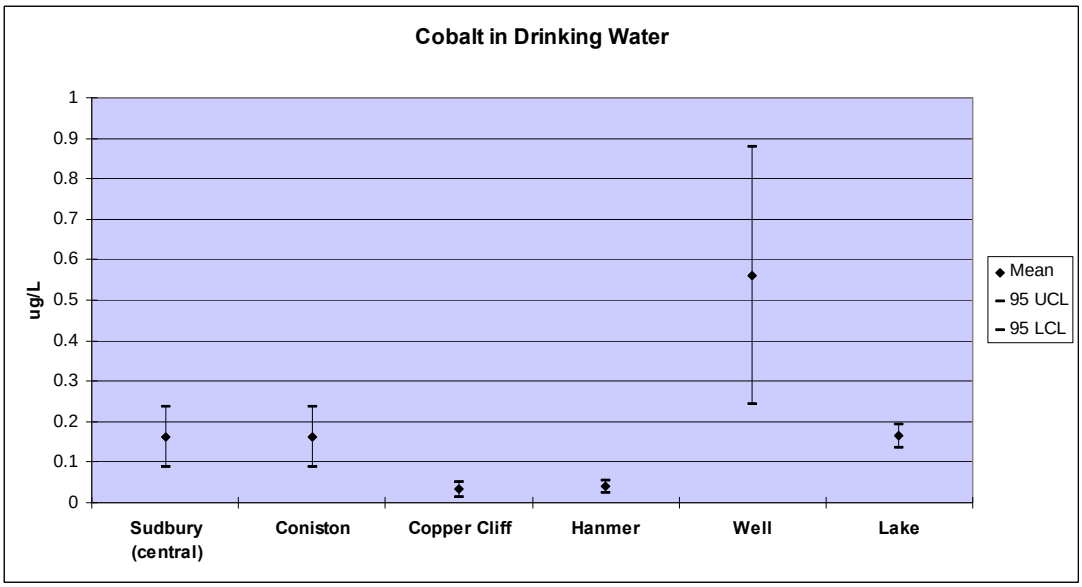


**Figure 4-11 Cobalt Concentrations in Drinking Water in the GSA (µg/L)**

It should be noted that cobalt was not part of the monitoring program in the Town of Falconbridge and therefore concentration data for this element are not available.

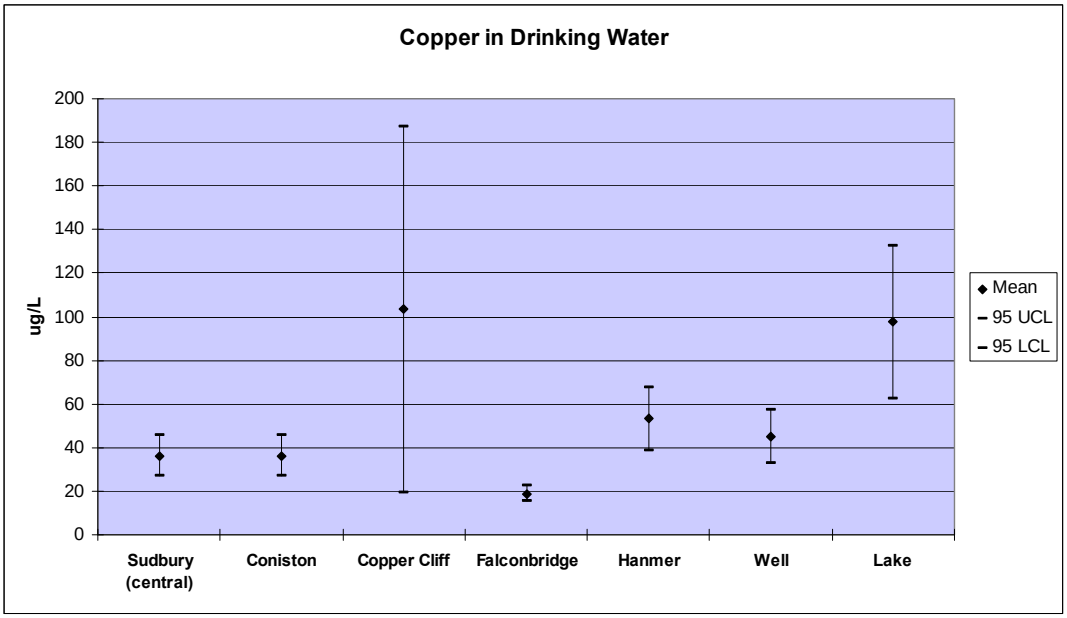


**Figure 4-12 Copper Concentrations in Drinking Water in the GSA (µg/L)**

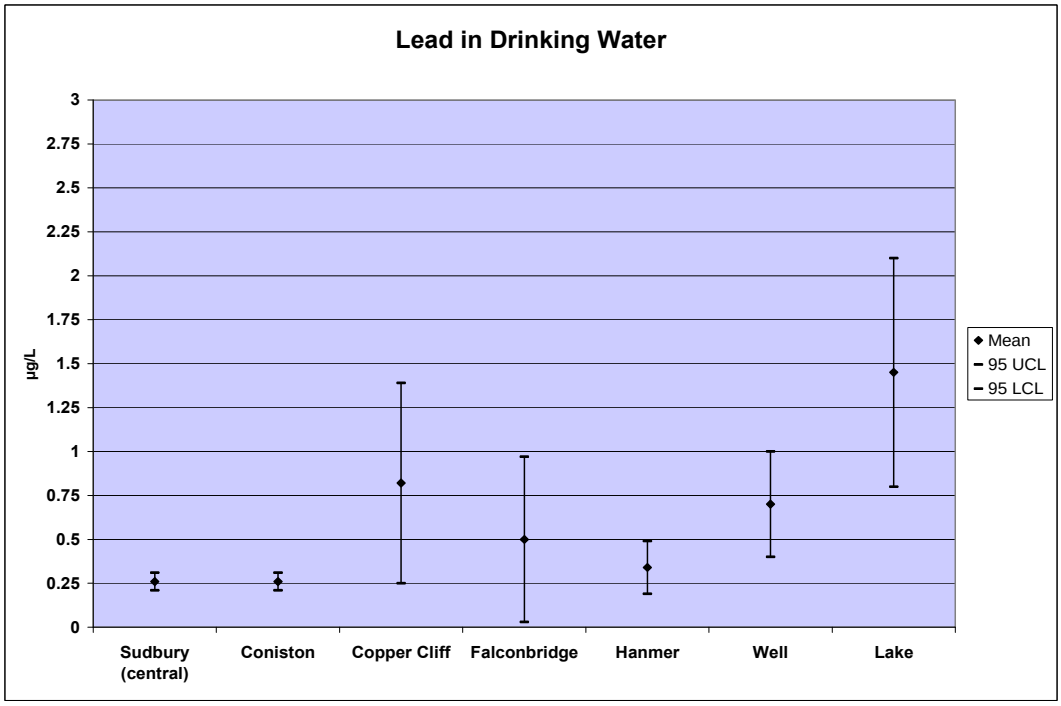


**Figure 4-13 Lead Concentrations in Drinking Water in the GSA (µg/L)**

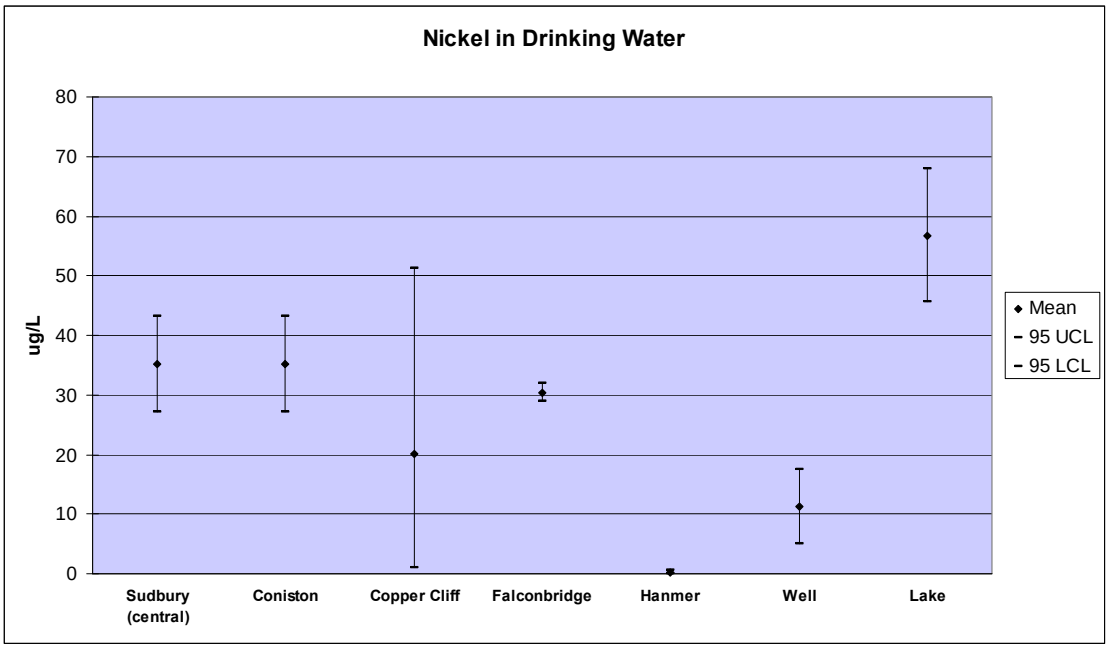


**Figure 4-14 Nickel Concentrations in Drinking Water in the GSA (µg/L)**

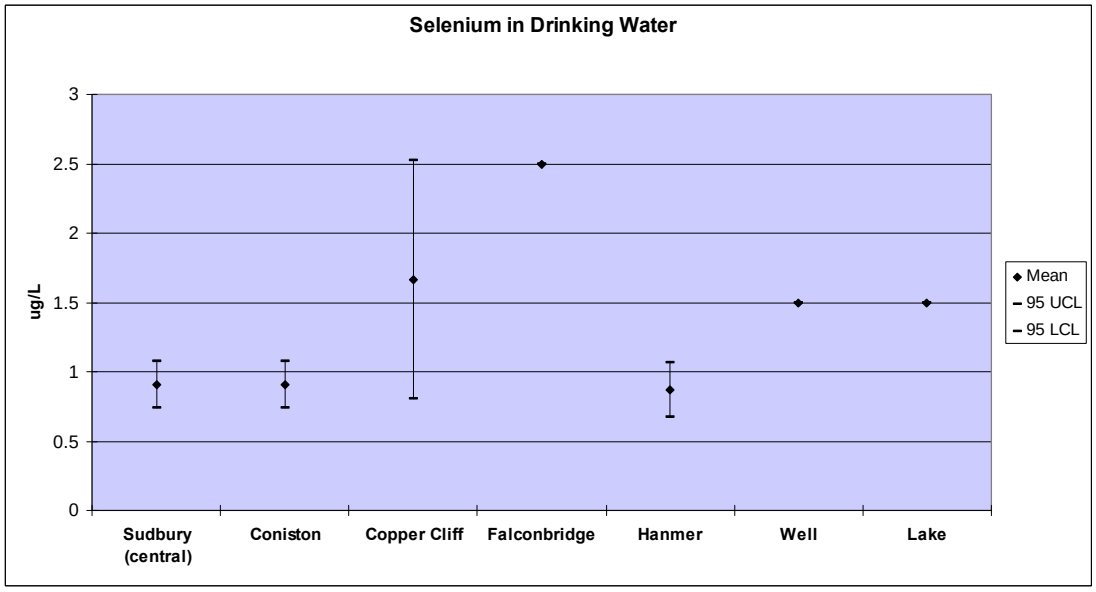


**Figure 4-15 Selenium Concentrations in Drinking Water in the GSA (µg/L)**

In general, COC concentrations in private drinking water supplies from both ground-water (well) and surface water (lake) sources in the GSA are within the range of drinking water concentrations measured in one of the five COI. In some cases, concentrations of arsenic and cobalt in well water, as well as nickel in lake water, were greater than those typically measured in municipal water supplies, but were still below the applicable regulatory guidelines.

By law, residents in each COI are required to use municipal drinking water supplies, where available, and are not permitted to construct private well systems on residential property except by permit. Participants in this survey and those using well or lake water as sources of drinking water are primarily located at the outskirts or completely outside of the COI being evaluated as part of this study. As well, they were also often at some distance from the smelters. Results of the soil sampling have indicated that soil concentrations decrease with distance from the smelters.

The results of the Drinking Water Survey are important to ensure exposures to residents using private drinking water supplies are considered; however, COC concentrations in the municipal drinking water supply were selected for estimating exposure of individuals through the consumption of tap water because these circumstances represent the majority of the population in the GSA (88%), the similarity of the private and municipal data, and because of the proximity of the municipal water consuming households to one of the three smelter operations. The uncertainty related to this assumption is discussed further in Chapter 7 of this volume.

#### 4.1.1.5 Vegetable Garden Produce Concentrations

Consumption of locally-grown vegetables, fruits and wild berries, specifically blueberries, is common in the GSA. Commercial farms and backyard gardens supplement the total daily intake of fruits and vegetables for GSA residents every year. In response to concerns raised by residents of the GSA regarding exposure to COC through the consumption of home-grown and locally-grown garden vegetables, fruits and wild berries, a vegetable garden survey was conducted across the GSA to collect Sudbury-specific COC concentration data for each of these food types. The study involved the collection of vegetable, fruit and crop samples from residential and commercial gardens across the GSA, wild blueberry and wild mushroom samples from remote areas, as well as co-located soil samples from each sample site. A brief summary of the vegetable garden survey can be found in Chapter 3 (see Section 3.7) of this report. The complete 2003 Vegetable Garden Survey Data Report can be found in Appendix E of this volume.

Locally-grown produce was divided into three categories: 1) aboveground vegetables; 2) below ground vegetables; and, 3) fruits. Wild blueberries and wild mushrooms were considered as separate food types because they are typically found in the wild, rather than in residential or commercial gardens. For the current assessment, Sudbury-specific COC concentration data collected from residential gardens in the COI were used to calculate 95% UCLM concentration values for each COC in both above ground and below ground vegetables in each of the COI. Due to the small number of fruit samples collected from residential gardens within the COI (n=4), all fruit samples collected in a COI garden were grouped together to calculate one 95% UCLM value for each COC across all COI. In addition, the exposure of a "local resident" was evaluated by assuming an individual could consume local vegetables and fruits from residential and commercial gardens across the entire GSA. Refer to Tables 3.31, 3.32, and 3.33 in Chapter 3 of this volume for a complete summary of the vegetable and fruit concentrations evaluated in the current assessment.

#### 4.1.1.6 Fish Tissue Concentrations

Angling and fishing are significant recreational activities in the Sudbury area and represent an important exposure pathway for human health. Consumption of locally caught fish by freshwater anglers and fisherman in the GSA may result in exposure to one or all of the COC being evaluated. To address this exposure pathway for human health, a fish tissue survey was conducted by the SARA Group in which fish, caught in eight lakes across the GSA, were analysed for various metals including arsenic, cobalt, copper, lead, nickel and selenium. A brief summary of the fish tissue survey can be found in Chapter 3 (see Section 3.8) of this report. The complete Metal Levels in Fish Tissues from Sudbury Lakes Data Report can be found in Appendix G of this volume.

COC concentrations measured in fillet (*i.e.*, muscle) tissue of collected fish were used to calculate a 95% UCLM for each COC. Forage fish and yellow perch less than 15 cm in length were excluded from this calculation as they do not represent fish typically caught for consumption by anglers. Table 3.27 in Chapter 3 provides a summary of the fish tissue concentrations for each COC.

#### 4.1.1.7 Indoor Dust Concentrations

Sudbury-specific indoor dust concentrations, along with co-located outdoor soil concentrations, were measured across the GSA as part of the Indoor Dust Survey conducted by the SARA Group in 2004. A brief description of the Indoor Dust Survey was provided in Chapter 3 (see Section 3.6) of this report. The complete Indoor Dust Survey Data Report is located in Appendix M of this volume.

The survey consisted of the collection of indoor dust from 91 residential homes located in one of the five COI and eight schools in the Rainbow District School Board. Co-located yard soil was collected concurrently from the front yards of residential homes participating in the dust survey to determine if there was a relationship between COC levels in indoor dust and outdoor soil in the GSA.

Initial review of the indoor dust and outdoor soil data indicated that indoor dust levels for each COC were 2.8 to 5.9 times higher than corresponding soil levels. It was also noted that as outdoor soil concentrations increased, soil appeared to become a more significant contributor to indoor dust concentrations of the COC. To most accurately describe this relationship, linear regression equations were developed for each COC to predict indoor dust concentrations as a function of outdoor soil concentrations.

It is noted that selenium was found at low levels in both yard soil and indoor dust. More than 38% of yard soil samples were below the reported minimum detection limit of 0.8 µg/g soil for selenium. Due to the large number soil samples which were reported to be less than the minimum detection limit, selenium was not evaluated in the regression analyses. Table 3.21 in Chapter 3 provides the best fit linear regression equations (*i.e.*, *ln*-transformed) based upon the paired outdoor soil and indoor dust concentration sets for the remaining five COC.

All regression equations were statistically significant and considered appropriate for the development of Sudbury-specific dust-to-soil relationships. These relationships were used to generate dust exposure values for the HHRA.

#### 4.1.1.8 Wild Game Tissue Concentrations

Limited monitoring data were available for metal concentrations in game meat in the study area; therefore, predicted concentrations were required for input to the human health exposure model to quantify potential exposures from this pathway. Few data are available which relate metal concentrations in soil and diet to metal concentrations in game meat, such as moose. Empirical data for metals are available for livestock (*i.e.*, cows) and small mammals. The following method used in the HHRA is recommended by the U.S. EPA (1999a) for predicting metal concentrations in mammals. The moose was selected as an appropriate wildlife receptor for consumption by humans based on the following:

- Moose require large home ranges and consume large amounts of forage;
- Residents of the GSA have confirmed that consumption of moose meat is common;

- Moose are predicted to have higher tissue metal concentrations based on the high consumption rates for forage;
- Moose are herbivores like the cows that were used to develop the empirical models to predict beef meat concentrations; and
- Moose consume grasses in the study area that were measured by field investigations for metal concentrations.

Moose are large mammals (body weight = 325 $\pm$ 59 kg) whose meat could potentially be impacted by COC through the consumption of impacted forage, aquatic plants, surface water and soil. Moose meat concentrations (in muscle tissues) were calculated following the U.S. EPA OSW (1998) methodology for predicting metal concentrations in beef cattle. For the purpose of estimating game tissue residue levels, wildlife was assumed to be exposed to chemicals through consumption of soil and food derived from the rural portion of the study area. Only the game meat concentrations predicted in Zone 2 of the ERA (see yellow zone outlined in Figure 4-16) were used for input to the human exposure model. Measured COC concentrations in soils from Zone 2 were the highest for the wild land areas and would provide a conservative estimate of game meat concentrations from other areas where concentrations are expected to be lower.

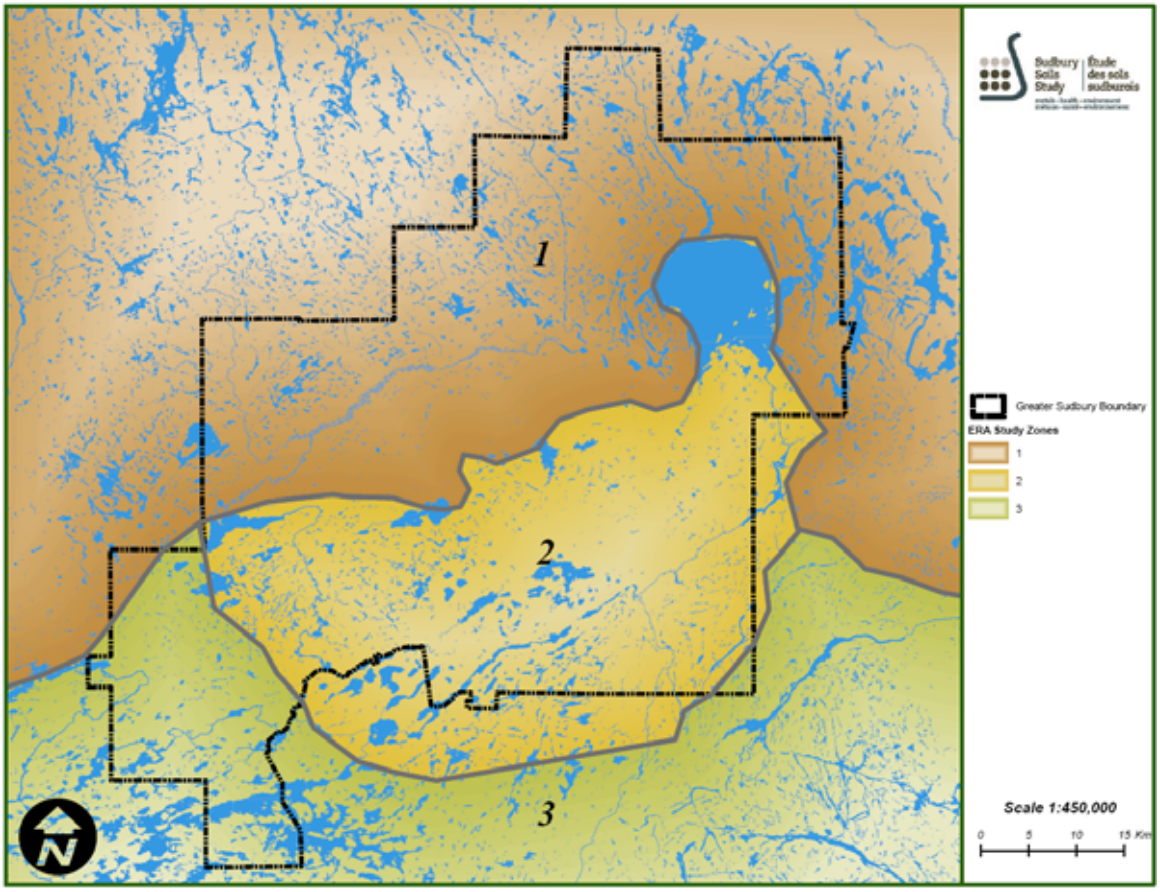


**Figure 4-16 Study Zones used by the Ecological Risk Assessment (ERA)**

The predicted moose meat concentrations were assumed to represent exposures that might be received by humans from other game such as deer and upland birds (*i.e.*, grouse). None of the COC are expected to biomagnify in the terrestrial food-chain; therefore, tissue concentrations for top predators were not predicted.

Table 4.6 provides a summary of the surface soil concentrations (0 to 5 cm depth) and Table 4.7 provides a summary of the soil concentrations (0 to 20 cm depth) in wild land Zone 2. The 95% upper confidence limit on the mean (95% UCLM) is also provided. A large number of duplicate samples were collected and analyzed within this dataset. The geometric means of these samples were calculated to represent the soil concentration at each location and depth and were used to calculate soil exposure concentrations (*e.g.*, the 95% UCLM).

**Table 4.6 Summary of Surface Soil Concentrations (ppm [n=168]) in Zone 2 (0 to 5 cm depth)**

| Metal | Min | Max | Mean | Median | 95 UCLM |
|---|---|---|---|---|---|
| **Arsenic** | 6.3 | 68.9 | 26.1 | 24.4 | 28.1 |
| **Cobalt** | 4.0 | 78.4 | 18.4 | 15.3 | 20.0 |
| **Copper** | 36.9 | 3830 | 453 | 397 | 495 |
| **Lead** | 3.9 | 145 | 56.4 | 56.1 | 59.7 |
| **Nickel** | 25.5 | 2900 | 434 | 317 | 477 |
| **Selenium** | 0.5 | 16.9 | 3.2 | 2.4 | 4.0 |

**Table 4.7 Summary of Soil Concentrations (ppm [n=168]) in Zone 2 (0 to 20 cm depth)**

| Metal | Min | Max | Mean | Median | 95 UCLM |
|---|---|---|---|---|---|
| **Arsenic** | 4.1 | 63.7 | 15.8 | 13.8 | 17.0 |
| **Cobalt** | 3.7 | 40.8 | 12.7 | 11.1 | 13.6 |
| **Copper** | 13.5 | 1510 | 252 | 208 | 276 |
| **Lead** | 3.6 | 103 | 34.1 | 27.8 | 37.7 |
| **Nickel** | 23.7 | 1240 | 245 | 195 | 279 |
| **Selenium** | 0.5 | 6.5 | 1.9 | 1.5 | 2.1 |

The distribution of COC in surface soil (*i.e.,* 0 to 5 cm depth) was used to estimate concentrations of metals in moose meat from direct soil ingestion and the distribution of COC in soil at depth (*i.e*., 0 to 20 cm depth) was used to estimate concentrations of metals in moose meat from forage ingestion. Two types of models were identified to estimate the potential distribution of metal concentrations in forage: 1) bioconcentration factor (BCF) models; and, 2) regression models. The following BCF equation was used to estimate concentrations:

$$C_i = C_s \times BCF_i$$

Where:

$C_i$ = Concentration of chemical in food "i" (mg/kg dw)

$C_s$ = Concentration of metal in soil (µg/g or ppm)

$BCF_i$ = Soil to food "i" bioconcentration factor ([mg chemical/kg dw] / [mg chemical/kg soil])

The following linear and transformed power-function regression models were also used to estimate concentrations in forage:

$$C_i = a + b \times C_s + E$$

Where:

| | | |
|---|---|---|
| $C_i$ | = | Concentration of chemical in food "i" (mg/kg dw) |
| a | = | Regression model intercept |
| b | = | Regression model slope |
| $C_s$ | = | Concentration of metal in soil (µg/g) |
| E | = | Regression model root mean square error |

The predictive empirical models provided in Table 4.8 were used with the distributions of soil concentrations in Table 4.7, above, to derive the distribution of forage concentrations that might be available for moose consumption. Table 4.9 provides a summary of the predicted concentrations that moose would be exposed to through consumption of forage in Zone 2.

**Table 4.8 Predictive Models Used to Estimate COC Concentrations in Shoots**

| COC | Model | Parameters | Basis |
|---|---|---|---|
| **Arsenic** | Linear regression | a=0.196; b=0.282; E=N(0,0.45) | Site-specific |
| **Cobalt** | BCF | LN(0.0507, 0.0559) | Site-specific |
| **Copper** | BCF | LN(0.0908, 0.132) | Site-specific |
| **Lead** | BCF | LN(0.105, 0.129) | Site-specific |
| **Nickel** | Linear regression | a=11.8; b=0.0845; E=N(0,11.1) | Site-specific |
| **Selenium** | Linear regression | a=0.256; b=0.235; E=N(0,0.365) | Site-specific |

Notes:
N – Normal distribution defined by (Mean, Standard Deviation)
LN – Log-normal distribution defined by (Mean, Standard Deviation)

**Table 4.9 Summary of Predicted Concentrations in Forage from Zone 2 (mg/kg dw)**

| Statistics | Arsenic | Cobalt | Copper | Lead | Nickel | Selenium |
|---|---|---|---|---|---|---|
| Mean | 0.64 | 0.55 | 18 | 3.6 | 33 | 0.71 |
| Median | 0.62 | 0.37 | 11 | 2.3 | 33 | 0.70 |
| Std. Deviation | 0.42 | 0.57 | 22 | 3.9 | 11 | 0.35 |
| Minimum | 0.0 | 0.03 | 0.29 | 0.15 | 0.0 | 0.0 |
| Maximum | 2.1 | 5.0 | 190 | 32 | 76 | 1.9 |
| 95$^{th}$ Percentile | 1.4 | 1.6 | 60 | 10 | 51 | 1.3 |

Concentrations of COC in surface water were represented by total metals measured in 30 lakes in the core area of the City of Greater Sudbury as part of the Urban Lakes study in 2003 (Co-Op, 2004). The complete surface water data set used in the moose exposure modelling is presented in Table 4.10.

**Table 4.10 COC in Surface Water (from Keller *et al*., 2004)**

| Lake | pH | As (µg/L) | Co (µg/L) | Cu (µg/L) | Pb (µg/L) | Ni (µg/L) | Se (µg/L) |
|---|---|---|---|---|---|---|---|
| Bennett | 6.74 | ***1*** | ***0.75*** | 9 | ***5.5*** | 24 | ***0.25*** |
| Bethel | 9 | ***0.25*** | ***0.75*** | 3 | ***5.5*** | 21 | ***0.25*** |
| Broder #23 | 6.38 | ***0.25*** | ***0.75*** | 10 | ***5.5*** | 49 | ***0.25*** |
| Brodill | 6.05 | ***0.25*** | ***0.75*** | 9 | ***5.5*** | 56 | ***0.25*** |
| Clearwater | 6.33 | ***0.25*** | ***0.75*** | 10 | ***5.5*** | 70 | ***0.25*** |
| Crooked | 5.78 | ***0.25*** | 2.4 | 35 | ***5.5*** | 108 | ***0.25*** |
| Crowley | 6.31 | ***0.25*** | ***0.75*** | 11 | ***5.5*** | 55 | ***0.25*** |
| Daisy | 6.2 | ***0.25*** | ***0.75*** | 12 | ***5.5*** | 80 | ***0.25*** |
| Dill | 6.61 | ***0.25*** | ***0.75*** | 10 | ***5.5*** | 49 | ***0.25*** |
| Forest | 6.18 | ***0.25*** | ***0.75*** | 12 | ***5.5*** | 91 | ***0.25*** |
| Grant | 7.21 | ***0.5*** | 1.9 | 5 | ***5.5*** | 53 | ***0.25*** |
| Hannah | 7.25 | ***0.5*** | ***0.75*** | 22 | ***5.5*** | 111 | ***0.25*** |
| Johnny | 6.76 | ***0.5*** | ***0.75*** | 19 | ***5.5*** | 85 | ***0.25*** |
| Laurentian | 6.53 | ***0.25*** | ***0.75*** | 14 | ***5.5*** | 37 | ***0.25*** |
| Linton | 6.16 | ***0.25*** | ***0.75*** | 10 | ***5.5*** | 59 | ***0.25*** |
| Little Raft | 7.02 | ***0.5*** | ***0.75*** | 8 | ***5.5*** | 38 | ***0.25*** |
| Lohi | 6.28 | ***0.25*** | ***0.75*** | 12 | ***5.5*** | 59 | ***0.25*** |
| Long | 7.1 | ***0.25*** | ***0.75*** | 12 | ***5.5*** | 47 | ***0.25*** |
| McFarlane | 7.33 | ***0.25*** | ***0.75*** | 8 | ***5.5*** | 51 | ***0.25*** |
| Middle | 6.91 | ***0.25*** | ***0.75*** | 24 | ***5.5*** | 114 | ***0.25*** |
| Minnow | 8.79 | ***0.5*** | ***0.75*** | 5 | ***5.5*** | 22 | ***0.25*** |
| Nepahwin | 7.4 | ***0.25*** | ***0.75*** | 11 | ***5.5*** | 45 | ***0.25*** |
| Raft | 6.61 | ***0.25*** | ***0.75*** | 12 | ***5.5*** | 74 | ***0.25*** |
| Ramsey | 7.43 | ***0.5*** | ***0.75*** | 12 | ***5.5*** | 55 | ***0.25*** |
| Richard | 7.25 | ***0.25*** | ***0.75*** | 8 | ***5.5*** | 57 | ***0.25*** |
| Robinson | 7.7 | ***1*** | ***0.75*** | 10 | ***5.5*** | 36 | ***0.25*** |
| Silver | 6 | ***0.25*** | 4.9 | 17 | ***5.5*** | 105 | ***0.25*** |
| St. Charles | 7.22 | ***0.25*** | ***0.75*** | 21 | ***5.5*** | 95 | ***0.25*** |
| Still | 7.55 | ***0.75*** | ***0.75*** | 15 | ***5.5*** | 58 | ***0.25*** |
| Tilton | 6.28 | ***0.25*** | ***0.75*** | 9 | ***5.5*** | 50 | ***0.25*** |

Values in italics and bolded represent 1/2 minimum detection limit.

A statistical summary of surface water data is provided in Table 4.11. Parameters were calculated using Microsoft Excel.

**Table 4.11 Statistical Summary of Surface Water Concentrations (µg/L)**

| Statistics | Arsenic | Cobalt | Copper | Lead | Nickel | Selenium |
|---|---|---|---|---|---|---|
| Mean | 0.37 | 0.98 | 13 | 5.5 | 62 | 0.25 |
| Median | 0.25 | 0.75 | 11 | 5.5 | 56 | 0.25 |
| Std. Deviation | 0.22 | 0.82 | 6.5 | 0 | 26 | 0 |
| Minimum | 0.25 | 0.75 | 3.0 | 5.5 | 21 | 0.25 |
| Maximum | 1 | 4.9 | 35 | 5.5 | 110 | 0.25 |
| 95% Upper Confidence Limit | 0.44 | 1.28 | 15 | NA | 71.21 | NA |

NA – Not applicable. The statistic could not be calculated based on the data set.

The complete data set for each COC was used to establish the distribution that most accurately characterizes the levels of each COC in Table 4.11 within surface water and to derive a 95% upper confidence limit on the mean (95% UCLM) that would apply to the entire study area. Table 4.12 shows the recommended distribution for each COC. Surface water quality was required in the moose exposure model to predict concentrations of metals in algae or aquatic vegetation.

**Table 4.12 Surface Water Assumptions Used for Input Variables**

| COC | Recommended Distribution | Parameters (µg/L) |
|---|---|---|
| **Arsenic** | Range | R(0.25, 1.0) |
| **Cobalt** | Normal | N(0.98, 0.82) |
| **Copper** | Log-Normal | LN(12.5, 14.97) |
| **Lead** | Constant = ½ Detection Limit | 5.5 |
| **Nickel** | Normal | N*(61.8, 69.96) |
| **Selenium** | Constant = ½ Detection Limit | 0.25 |

**Notes:**
Non-detect values were entered as ½ the detection limit.
R = Range distribution defined by (Minimum, Maximum); N = Normal distribution defined by (Mean, Standard Deviation); N* = Normal distribution defined by (Mean, 95% UCLM); and, LN = Log-normal distribution defined by (Mean, 95% UCLM)

The distribution of surface water quality provided in Table 4.12 was used to estimate algae or aquatic plant concentrations. Water-to-algae BCFs provided by U.S. EPA (1999a) were used to estimate the potential distribution of metal concentrations in algae and aquatic plants. The following equation was used to estimate algae concentrations:

$$C_{ap} = C_w \times BCF_{ap}$$

Where:

$C_{ap}$ = Concentration of chemical in aquatic plant (mg/kg dw)

$C_w$ = Concentration of dissolved metal in water (mg/L)

$BCF_{ap}$ = Water-to-aquatic-plant bioconcentration factor ([mg chemical/kg dw] / [mg chemical/L water])

The BCFs in Table 4.13 were used with the distributions of surface water concentrations in Table 4.12 to derive the distribution of aquatic plant concentrations for the moose exposure model (Table 4.14). In order to use the BCFs in the model, all values were converted from a wet weight basis to a dry weight basis by multiplying by a factor of 2.92 (U.S. EPA, 1999a). The conversion factor assumes moisture content of 65.7% for algae and aquatic plants. In addition, total metal concentrations were used because dissolved concentrations were not available for surface water. Therefore, using the BCFs listed in Table 4.13 will result in conservative estimates of metal concentrations in aquatic plants (Table 4.14).

**Table 4.13 Bioconcentration Factor (BCF) Assumptions Used for Estimating Aquatic Plant Concentrations (mg chemical / kg w.w.) / (mg chemical / L water)[a]**

| COC | Recommended Value (wet weight) | Value (dry weight) | Comment |
|---|---|---|---|
| **Arsenic** | 293 | 856 | Used three empirical values |
| **Cobalt** | 61 | 178 | No data available; assumed equal to nickel |
| **Copper** | 541 | 1,580 | Used recommended point |
| **Lead** | 1,706 | 4,982 | Used three empirical values |
| **Nickel** | 61 | 178 | Used four empirical values |
| **Selenium** | 1,845 | 5,387 | Used three empirical values |

[a] BCFs selected from U.S. EPA (1999a)

[b] Nickel data was used to characterize the BCF for cobalt due to lack of available data for cobalt and the proximity of the two COC within the Periodic Table. Data for uptake of cobalt from water into algae was available, with BCFs in the range of 150 to 3000. The Ni BCF of 178 is within this range. The geometric mean of eight BCFs for cobalt nitrate into green algae was 430 (3 week exposure).

**Table 4.14 Predicted Distribution of Concentrations in Aquatic Plants (mg/kg dw)**

| Statistics | Arsenic | Cobalt | Copper | Lead | Nickel | Selenium |
|---|---|---|---|---|---|---|
| Mean | 0.55 | 0.20 | 6.7 | 27 | 11 | 1.4 |
| Median | 0.54 | 0.19 | 0.84 | 27 | 11 | 1.4 |
| Std. Deviation | 0.19 | 0.12 | 35 | 0.0 | 0.88 | 0.0 |
| Minimum | 0.21 | 0.0 | 0.0 | 27 | 8.0 | 1.4 |
| Maximum | 0.86 | 0.59 | 820 | 27 | 13 | 1.4 |
| 95$^{th}$ Percentile | 0.83 | 0.42 | 24 | 27 | 13 | 1.4 |

The consumption of terrestrial and aquatic plants and from consumption of surface water and soil was used to estimate game meat concentrations through the use of biotransfer factors (BTFs) as recommended by the U.S. EPA OSW (1998). The BTFs (Table 4.15) are developed based on empirical studies with beef cattle and are used to translate the estimated daily dose of a chemical to a tissue concentration. The following equation was used to estimate moose meat concentrations:

$$C_M = BTF \times \left( EDI_F + EDI_S + EDI_W \right)$$

Where:

| | | |
|---|---|---|
| $C_m$ | = | Concentration of chemical in moose meat (mg/kg fw) |
| BTF | = | Bio transfer factor (days/kg fw) |
| $EDI_F$ | = | Estimated daily intake from terrestrial & aquatic plants (mg-chemical/day) |
| $EDI_S$ | = | Estimated daily intake from soil (mg-chemical/day) |
| $EDI_W$ | = | Estimated daily intake from water (mg-chemical/day) |

**Table 4.15 Biotransfer Factors (BTFs) Used to Predict Game Meat Concentrations (days/kg-f.w.)**

| Chemical | Value | Distribution | Reference/Comment |
|---|---|---|---|
| **Arsenic** | 0.002 | Static | Baes *et al.*, 1984 Cited In: U.S. EPA OSW, 1998 |
| **Cobalt** | 0.02 | Static | Baes *et al.*, 1984 Cited In: U.S. EPA OSW, 1998 |
| **Copper** | 0.01 | Static | Baes *et al.*, 1984 Cited In: U.S. EPA OSW, 1998 |
| **Lead** | 0.0003 | Static | Baes *et al.*, 1984 Cited In: U.S. EPA OSW, 1998 |
| **Nickel** | 0.006 | Static | Baes *et al.*, 1984 Cited In: U.S. EPA OSW, 1998 |
| **Selenium** | 0.440 | U(0.00227,1.13) | Used range observed between beef, pork and chicken (U.S. EPA OSW, 1998) |

U: Uniform distribution assumed; U(Minimum, Maximum)

The predicted game meat concentrations used in the HHRA are presented in Table 4.16.

**Table 4.16 Predicted Concentrations of COC in Moose Meat (mg/kg fw)**

| Statistics | Arsenic | Cobalt | Copper | Lead | Nickel | Selenium |
|---|---|---|---|---|---|---|
| Mean | 0.004 | 0.04 | 0.62 | 0.004 | 0.60 | 1.28 |
| Median | 0.003 | 0.02 | 0.32 | 0.002 | 0.51 | 0.88 |
| Std. Deviation | 0.004 | 0.05 | 1.03 | 0.005 | 0.38 | 1.30 |
| Minimum | 0.0000 | 0.0008 | 0.007 | 0.0003 | 0.004 | 0.002 |
| Maximum | 0.04 | 0.60 | 16.2 | 0.07 | 3.08 | 9.39 |
| 95$^{th}$ Percentile | 0.004 | 0.04 | 0.68 | 0.004 | 0.62 | 1.36 |

### 4.1.2 Background Exposure Assessment

The COC considered in the current HHRA are naturally present within the environment, and/or have a number of anthropogenic sources, which are not associated with historic or ongoing emissions from the existing smelting operations. As such, everyone is exposed to a variety of chemicals from a number of sources on a daily basis, regardless of where they live. It is, therefore, important to consider "background" exposures and risks in an HHRA to determine the extent to which residents are more exposed to chemicals from their environment than would normally be expected in the absence of a major source of COC, such as smelting emissions.

Background or baseline exposures are defined as exposures to chemicals that are not related to the point source or area of impact under assessment. Background sources may be either naturally-occurring or anthropogenic (human-made) and contribute to levels of contaminants in foods, water, air, soil, and consumer products that humans are commonly exposed to everyday. Background exposures can occur outside of the area of concern or at other time periods than those defined by the assessment.

The purpose of conducting a background exposure assessment is to determine the contribution of background sources to an individual's total exposure. The remaining proportion of total exposure, after accounting for background exposures can in part be attributed to exposures originating from historical and current smelting activities in the GSA. The contributions of these two sources of exposure to the COC are considered in the derivation of soil clean-up criteria.

Therefore, an additional exposure and risk assessment was conducted to evaluate the degree of exposure of the receptors to the COC without the contribution of the Vale Inco and Xstrata Nickel smelters. In this case, such an assessment provides an indication of the exposures experienced by a typical Ontario resident (TOR), based on ambient or background concentrations in water, air, soil, dust, and food sources. Predicted TOR exposures can then be compared with the exposures attributed to smelting activity, to give an indication of total exposure to COC from all known sources. In addition to using background exposure to account for an individual's estimated total daily intake, background assessments can also be used as benchmarks of comparison that aid in determining the significance of the exposures from the study area relative to typical Ontario background exposures. Such relative contribution analysis can be useful in putting exposure and risk estimates into perspective, and guiding the development of risk management recommendations (*e.g.,* if study area exposures and risks are estimated to be less than or similar to typical Ontario exposures, the need for risk management measures may be reduced or become unnecessary). Evaluation of typical background exposures also assists in the interpretation and validation

of predictive modelling data, which increases stakeholder confidence in the overall results of the HHRA process.

Background COC concentrations used in the current background exposure assessment were derived from monitoring programs in Ontario and across Canada. The background COC concentrations, described in more detail below, were used to calculate 95% UCLM values in outdoor air, soil and drinking water for a Typical Ontario Resident (TOR) scenario.

#### 4.1.2.1 Data Used in Exposure Assessment for Typical Ontario Residents

It is important to characterize background sources of exposure to COC when conducting a detailed HHRA. By incorporating background sources of exposure into the assessment, total estimated exposure from all sources (including background) can be compared to the reference exposure value (*e.g.,* a tolerable daily intake (TDI) or reference dose (RfD)), without needing to make decisions about how the TDI or RfD value should be reduced to account for exposures that were not explicitly evaluated. For example, for compounds considered to act *via* a threshold mechanism of toxicity, the entire RfD or TDI can be compared to the site-specific estimated exposure rate, rather than employing an allocation factor. Secondly, a good understanding of background exposures expected for the TOR can be useful as a contextual framework within which to consider site-related exposures.

The exposure equations used for the typical Ontario background assessment are the same as those used to calculate exposure rates of individuals in the GSA. However, it should be noted that the data used to evaluate TOR exposures has been derived from a variety of generic sources, and the TOR receptor scenario, on its own, would not be appropriate for use in assessing potential health risks to the typical Ontario resident. It is simply included in the current assessment to provide a rough comparison point for the evaluation of the Sudbury-specific health risk predictions, and to place them into an overall Ontario context.

***Background Outdoor Air Concentrations***

Ontario Air Quality reports were evaluated as a source of background air chemical data for Ontario. Under the Ontario Ministry of the Environment's (MOE) air quality monitoring program, sulphur dioxide, ozone, nitrogen dioxide, total reduced sulphur compounds, carbon monoxide and fine particulate matter are monitored in air at select Ontario locations. Certain metals in particulate matter are also monitored. Annual statistics that combine data for all the monitoring sites in Ontario are readily available for the year 2002 and earlier; however, of the COC for the HHRA, only cobalt, lead and nickel are monitored under

this program. It should be noted that selection of monitoring sites for this program was biased toward locations with actual or perceived air quality issues; therefore, summary statistics from the Ontario Air Quality reports do not necessarily represent typical Ontario conditions. Refer to Section 3.1.2 for a discussion of the limited data available for these metals.

The National Air Pollution Surveillance (NAPS) network monitors air pollutants at monitoring stations across Canada. The NAPS data provide a long-term archive of air pollution data at urban and rural locations in all regions of Canada. All six COC are among the metals analyzed in particulate samples from NAPS monitoring stations.

The NAPS dataset was selected as background outdoor air concentrations for the TOR scenario because it provides the advantage of consistency across chemicals, regions and time periods. The 2002 data from a monitoring station in a residential area of Toronto (185 Judson Street) provided the most robust set of data and was selected to represent background air concentrations in Ontario. However, as only 53 samples were available, there was insufficient information on which to generate a 95% UCLM for each COC. As such, Table 4.17 provides the arithmetic mean for each COC which where used in the current assessment (Dann, 2005 pers. comm.).

**Table 4.17 Typical Ontario Ambient Air Concentrations (µg/m$^3$) (185 Judson Street, Toronto)**

| COC | No. of Samples | Arithmetic Mean of $PM_{10}$ samples |
|---|---|---|
| **Arsenic** | 53 | 0.001 |
| **Cobalt** | 53 | 0.002 |
| **Copper** | 53 | 0.009 |
| **Lead** | 53 | 0.008 |
| **Nickel** | 53 | 0.001 |
| **Selenium** | 53 | 0.002 |

***Background Soil Concentrations***

A data set of background soil concentrations collected by the Geological Survey of Canada in 1994 during a regional geochemical survey of Canada, was reviewed for the current assessment. The objective of the survey was to define the range of background concentrations of metals in the surface soils of different eco-regions of Canada. Summary statistics of COC concentrations in surface soils of seven eco-regions in Southern Ontario were reviewed (Garrett, 2005 pers. Comm.). However, raw data were not available. Based on the available dataset, it was not possible to derive a background value for all of

Southern Ontario, or to exclude concentrations measured in mineralized soils. Therefore, these data were not used in estimating background soil concentrations for the current assessment.

Ontario Typical Ranges (OTRs) represent the expected range of concentrations of contaminants in surface soil from areas in Ontario not subjected to the influence of known point sources of emissions. The OTR report (OMEE, 1994) presents ranges, means and the 98$^{th}$ percentile soil concentration ($OTR_{98}$) for each of the COC. The $OTR_{98}$ represents an upper limit of normal concentrations in Ontario. These values apply to the land use and soil type in Ontario for which they were developed.

More recently, the MOE (2004) derived background values, based on the OTR values, intended to represent the upper limits of typical province-wide background concentrations that are not contaminated by point sources. These values (see Table 4.18) were extracted from Table 1 of the Record of Site Condition Regulation (*O. Reg.* 153/04) and used as the soil concentrations for the TOR scenario.

**Table 4.18 Typical Ontario Soil Concentrations (µg/g)**

| COC | Background Value (*O Reg.* 153/04 - Table 1) |
|---|---|
| Arsenic | 17 |
| Cobalt | 21 |
| Copper | 85 |
| Lead | 120 |
| Nickel | 43 |
| Selenium | 1.9 |

It should be noted that use of these regulatory benchmarks as comparison points to the Sudbury-specific soil concentrations is very conservative, as they represent 98$^{th}$ percentiles of a dataset of soil concentrations collected across Ontario (excluding Sudbury), and not the 95% UCLM. Unfortunately, as noted previously, the raw dataset was unavailable to allow the calculation of the 95% UCLM, and as such, only the mean or 98$^{th}$ percentile of the data could potentially be selected to represent TOR soil concentrations. The SARA Group considered the use of the mean soil concentration was not an appropriate statistic to represent a reasonable upper bound estimate of soil concentrations. As such, though more conservative, the 98$^{th}$ percentile statistic was selected for the current assessment.

***Background Drinking Water Concentrations***

To estimate exposure of a TOR to COC in drinking water, monitoring data from over 170 water treatment plants and well supplies across Ontario were used to calculate 95% UCLM values for each COC. Drinking water monitoring data were provided by the MOE based on information collected by the DWSP, a voluntary reporting system operated by the MOE in cooperation with municipalities across the province

(MOE, 2005a). For the purpose of estimating exposure to COC concentrations in typical drinking water under a TOR scenario, samples collected from distribution systems from 1997 through 2002 were included in the calculation. These samples were unfiltered and were analysed for total metal concentrations. Raw and treated water samples were not included in the estimate of exposure to COC in drinking water because they are typically collected before circulation through the water supply system; and therefore, do not accurately reflect drinking water that would be consumed by a TOR in a residential scenario (*i.e.,* from a kitchen faucet).

Drinking water concentrations reported by the DWSP are available to the public; however, these concentrations must be interpreted with caution. In some cases, negative concentration values were reported in the DWSP data due to corrections made to the analytical results based on blanks or interferences. Concentration values of zero were also reported. For the purpose of calculating a 95% UCLM for COC concentrations in drinking water, concentration values were replaced with a value equal to half the minimum detection limit (MDL) where negative or zero values occurred.

A summary of the estimated 95% UCLM COC concentrations in drinking water for a TOR exposure scenario are presented in Table 4.19.

**Table 4.19 Typical Ontario Drinking Water Concentrations (µg/L) (Drinking Water Surveillance Program Reports - MOE, 2005a)**

| COC | No. of Samples | Min | Max | Mean | 95% UCLM |
|---|---|---|---|---|---|
| **Arsenic** | 2296 | 0.03 | 15.0 | 0.56 | 0.64 |
| **Cobalt** | 2296 | 0.0023 | 2.85 | 0.08 | 0.09 |
| **Copper** | 2296 | 0.0025 | 0.00126 | 32.5 | 40.8 |
| **Lead** | 2301 | 0.0074 | 331 | 0.91 | 1.89 |
| **Nickel** | 2296 | 0.0006 | 113 | 1.41 | 2.18 |
| **Selenium** | 2291 | 0.0086 | 19.9 | 1.42 | 1.58 |

### 4.1.3 Market Basket Estimated Daily Intakes

Food represents a critical pathway of exposure to the COC for the residents of the GSA. Foods consumed and purchased from grocery stores, supermarkets, butchers, *etc,* are considered background sources of exposure and contribute to an individual's total level of exposure to COC. The exposures to COC through the consumption of store-bought foods is termed the *market basket estimated daily intake* or EDI. As part of the HHRA, a literature review was conducted to obtain published data on the concentrations of COC in store-bought foods (*i.e.,* supermarket or market basket food items) which Sudbury residents may

be consuming. The details of this literature review and the methodology used to estimate an individual's daily intake from market basket sources can be found in Appendix D of this volume.

An EDI is defined as the estimated daily intake of a chemical that is unrelated to any specific contaminated site (*i.e.*, normal "background" exposure) (CCME, 2005). It is characterized by an average Canadian's exposure to low levels of chemicals commonly found in air, water, food, soil, and consumer products (CCME, 2005). A market basket EDI ($EDI_{MB}$) is defined as the estimated daily intake of a chemical that is related to food commonly purchased in the supermarket and other points of purchase (*e.g.,* bakery, butchery), prepared, and consumed by urban Canadians.

The purpose of the $EDI_{MB}$ is to incorporate background exposure when characterizing an individual's exposure to COC. This is to ensure that a portion of a chemical's TDI is apportioned to background sources such that the total exposure to background levels, plus soil concentrations at the acceptable benchmark level do not exceed the TDI (CCME, 2005). In the context of the HHRA, the purpose of the $EDI_{MB}$ is two fold:

1. To ensure that background sources are included in the exposure assessment of Sudbury residences; and
2. To ensure that background sources are accounted for when calculating a Sudbury-specific soil risk management.

The purpose of the literature review was to identify the most appropriate food data to characterize Sudbury area residents' background exposure to store-bought foods. In Canada, most supermarkets foods are from sources distributed across North America and are generally not specific to the location of the supermarket. Thus, food purchased in Sudbury should resemble the foods purchased in other cities in Canada, particularly those in Ontario. An exception is the locally grown fruit and vegetables that are seasonally available in Sudbury. These were assessed separately and explicitly incorporated in the exposure assessment.

The purpose of the current *market basket* review was threefold: i) identify the key food item categories making up the diet of Sudbury residents; ii) determine the estimated daily intake rates for each food category; and, iii) determine the range of COC concentrations in each food category. The information generated from this phase of the study was incorporated into the exposure pathway model of the HHRA as the Estimated Daily Intake (EDI) for each COC.

The food concentrations used in the derivation of the $EDI_{MB}$ were based on the most applicable data available for food purchased in a Canadian supermarket. Food concentrations were calculated as the 95% UCLM.

In order to determine the most appropriate data to use in the Sudbury HHRA, the following criteria were used:

- Food concentration data were Canadian-specific (if Canadian data were unavailable, the literature search extended to international studies, preferably American);
- Food was purchased from a supermarket or other public point-of-purchase (*e.g.*, bakery, butcher, *etc.*);
- Food was prepared and/or cooked for normal consumption;
- Data were reported with adequate summary statistics (raw data, or at a minimum, the sample number, mean concentration and range);
- The minimum detection limits were adequately low to detect the metal in most of the food items; and,
- The quality of the study design and the comprehensiveness of the data collected were considered appropriate for use in this HHRA.

The databases selected for calculating the $EDI_{MB}$ are summarized in Table 4.20 (refer to Appendix D for the complete datasets).

**Table 4.20 Summary of Databases Selected for Use in the Development of the $EDI_{MB}$**

| COC | Location | Date | Description | Reference |
|---|---|---|---|---|
| As | Six Canadian cities | 1985 and 1988 | Canadian Total Diet Study[1]: Total As analyzed in supermarket foods | Dabeka *et al.*, 1993 |
| Co | Eight Canadian cities | 1993 to 1999; and 2000; 2002 | Canadian Total Diet Study[1]: Total Co analyzed in supermarket foods, Supplemented with green leafy vegetable data from Port Colborne | Health Canada, 2004a; Dabeka and McKenzie, 2005 pers. comm.; JWEL, 2004a |
| Cu | Eight Canadian cities | 1993 to 1999 and 2000 | Canadian Total Diet Study[1]: Total Cu analyzed in supermarket foods | Health Canada, 2004a; Dabeka and McKenzie, 2005 pers. comm. |
| Ni | Port Colborne | 2002 | Total Ni analyzed in foods from local supermarkets, food outlets, butchers eateries, and markets[2] | JWEL, 2004a |
| Pb | Canada | 2000 | Canadian Total Diet Study[1]: Total Pb analyzed in supermarket foods | Dabeka and McKenzie, 2005 pers. comm. |
| Se | United States | 1991 to 2002 | U.S. FDA Total Diet Study[3]: Total Se analyzed in supermarket foods | U.S. FDA, 2004 |

[1] All non-detected food concentrations were assumed by the authors to be the full detection limit.
[2] All non-detected food concentrations were assumed to be ½ the detection limit.
[3] All non-detected food concentrations were assumed to be ½ the detection limit.

For the purposes of applying the food concentrations to the $EDI_{MB}$, the raw data were obtained for all the datasets; non-detect data points were assigned a value of one half of the detection limit and the 95% UCLM of the food categories was calculated. The data used in the derivation of the $EDI_{MB}$ are summarized in Appendix D.

A brief summary of each COC is provided herein. For more detail, refer to Appendix D.

***Arsenic***

There were a number of Canadian market basket surveys available for arsenic (JWEL, 2004a; Dabeka *et al.,* 1993; MOE, 1987). Some of the market basket studies analyzed total arsenic (*e.g*., JWEL, 2004a; Dabeka *et al.,* 1993), while others analyzed both total and inorganic forms (MOE, 1987).

The database selected for use in the Sudbury HHRA was the Dabeka *et al*. (1993) Canadian Total Diet Study (CTDS) because it fulfilled all of the selection criteria and was found to be the most appropriate for arsenic. In this survey, food was sampled from supermarkets in six Canadian cities[1] and prepared as for normal consumption by Canadians (Dabeka *et al*., 1993). Raw data and summary statistics were available and the detection limits were appropriate, ranging from 0.3 to 1.1 ng/g wet weight. Unfortunately, arsenic was not analyzed in the Canadian TDS data for the period 1993 to 1999, and 2000 due to limited government resources (Dabeka and McKenzie, 2005 pers. comm.). Therefore, the available data are greater than 10 years old.

The more recent Port Colborne database (*i.e*., JWEL, 2004a) was not selected because it had inappropriately high detection limits (*i.e*., arsenic was non-detectable in 97% of food samples; detection limit was ~50 ng/g dw [~10 ng/g ww for vegetables[2]]); resulting in highly uncertain estimates of food concentrations. For that analysis, non-detectable arsenic concentrations were assumed to be equal to half the detection limit (JWEL, 2004a), an assumption that is typically conservative. This may explain why the mean concentrations for the food categories in the JWEL (2004a) data are consistently higher than those in the Dabeka *et al*. (1993) study. Due to a lack of any alternatives, the Port Colborne data were used for arsenic concentrations in infant formula.

[1] The six Canadian cities where food was sampled are: Ottawa (sampled twice), Halifax, Winnipeg, Vancouver and Toronto (Dabeka *et al*., 1993). The authors report no significant differences in the arsenic levels in food items between the cities where the food was collected (Dabeka *et al*., 1993). (Surveys of market basket foods are generally considered to be nationally representative because the foods sampled tend to be nationally distributed.) For this reason, data from multiple cities can be combined to create a larger and more robust database.

[2] Calculated for illustrative purposed only, and assumes 80% moisture content for vegetables.

Many studies concerned with estimating the dietary intake of arsenic have traditionally been based on surveys of total arsenic in food, including both organic and inorganic forms of arsenic. According to Schoof *et al*. (1999), arsenic concentrations in food were dominated by the relatively non-toxic organic forms of arsenic found in seafood. Schoof *et al.* (1999) conducted a market basket survey of inorganic arsenic in 40 different commodities which were anticipated to provide approximately 90% of the dietary intake of inorganic arsenic. Four samples of each commodity were collected and, analyzed for total and inorganic arsenic. Total arsenic was analyzed using a NaOH digestion and ICP-MS while inorganic arsenic was analyzed using a HCL digestion and hydride AAS. The results provided by Schoof *et al*. (1999) were consistent with other studies, in that total arsenic concentrations among seafood products were highest; however, inorganic arsenic concentrations observed in seafood were not elevated and ranged between less than 1 ng/g to 2 ng/g. According to Schoof *et al.* (1999), raw rice was found to have the highest inorganic arsenic content among all food commodities tested.

The arsenic concentration data used to establish estimated daily intake rates of inorganic arsenic from market basket and local foods (*e.g.,* home garden vegetables, *etc*.) were based on total arsenic measurements (*i.e.,* organic plus inorganic species). As a result, total arsenic concentrations reported for various food groups had to be corrected by the fraction of total arsenic that is present as inorganic species. The Schoof *et al*. (1999) data provided mean concentrations of total and inorganic arsenic in 40 different food commodities. From these data, the fraction of total arsenic which is inorganic could be derived for each food group. As previously indicated, at least four different food types within each commodity were analyzed for total and inorganic arsenic and, therefore, the ratios of inorganic over total arsenic content were developed for each food group within each commodity. The arithmetic mean ratio of different food groups was used to adjust the total arsenic concentration of a particular food group to an inorganic arsenic concentration. Table 4.21 provides data from Schoof *et al*. (1999) that were used to calculate the mean fraction of inorganic arsenic in different food groups.

**Table 4.21 Fraction of Inorganic Arsenic in Various Food Groups**

| Food Group | Total As (ng/g) | Inorganic As (ng/g) | Fraction Inorganic |
|---|---|---|---|
| ***Fats, oils, sweets, nuts*** | | | |
| Beet sugar | 12.2 | 3.50 | 0.29 |
| Cane sugar | 23.8 | 4.40 | 0.18 |
| Corn syrup | 6.00 | 0.40 | 0.07 |
| Butter | 1.80 | 1.10 | 0.61 |
| Soybean oil | 1.80 | 1.10 | 0.61 |
| Salt | 4.80 | 0.80 | 0.17 |
| Beer | 2.70 | 1.80 | 0.67 |

**Table 4.21 Fraction of Inorganic Arsenic in Various Food Groups**

| Food Group | Total As (ng/g) | Inorganic As (ng/g) | Fraction Inorganic |
|---|---|---|---|
| Peanut butter | 43.6 | 4.70 | 0.11 |
| MEAN VALUE | 12.1 | 2.23 | 0.34 |
| ***Milk, yogurt, cheese*** | | | |
| Milk, skim (non-fat) | 2.60 | 1.00 | 0.38 |
| Milk, whole | 1.80 | 1.00 | 0.56 |
| MEAN VALUE | 2.20 | 1.00 | 0.47 |
| ***Meat, poultry, eggs*** | | | |
| Beef | 51.5 | 0.40 | 0.01 |
| Chicken | 86.4 | 0.90 | 0.01 |
| Pork | 13.5 | 0.60 | 0.04 |
| Eggs | 19.9 | 1.00 | 0.05 |
| MEAN VALUE | 42.8 | 0.73 | 0.03 |
| ***Vegetables*** [1] | | | |
| Beans | 2.10 | 1.20 | 0.57 |
| Carrots | 7.30 | 3.90 | 0.53 |
| Corn | 1.60 | 1.10 | 0.69 |
| Cucumber | 9.60 | 4.10 | 0.43 |
| Onions | 9.60 | 3.30 | 0.34 |
| Potatoes | 2.80 | 0.80 | 0.29 |
| Tomato | 9.90 | 0.90 | 0.09 |
| MEAN VALUE | 6.13 | 2.19 | 0.42 |
| ***Fruit*** [2] | | | |
| Apple, raw | 4.80 | 1.80 | 0.38 |
| Apple, juice | 7.60 | 2.80 | 0.37 |
| Bananna | 2.30 | 0.60 | 0.26 |
| Grapes | 10.2 | 3.60 | 0.35 |
| Grape Juice | 58.3 | 9.20 | 0.16 |
| Orange Juice | 4.80 | 1.00 | 0.21 |
| Peaches | 3.40 | 2.30 | 0.68 |
| Watermelon | 40.2 | 8.90 | 0.22 |
| MEAN VALUE | 16.5 | 3.78 | 0.33 |
| ***Bread*** | | | |
| Corn (meal) | 38.6 | 4.40 | 0.11 |
| Flour | 39.1 | 10.9 | 0.28 |
| Rice | 303 | 73.7 | 0.24 |
| MEAN VALUE | 127 | 29.7 | 0.21 |
| ***Fish*** | | | |
| Saltwater finfish - mean (n=4) | 2360 | 0.50 | 0.0002 |
| Canned Tuna - mean (n=4) | 512 | 1.00 | 0.002 |
| Shrimp - mean (n=4) | 1890 | 1.90 | 0.001 |
| Freshwater finfish - mean (n=4) | 160 | 1.00 | 0.006 |
| MEAN VALUE | 1230 | 1.10 | 0.002 |

[1] Three (3) samples were excluded from the vegetable category since the inorganic arsenic content of these 3 samples were reported to be greater than their corresponding total arsenic contents.

[2] One (1) sample was excluded form the fruit category since this sample indicated that the inorganic arsenic content was greater than the total arsenic content.

***Cobalt***

A number of Canadian market basket studies are available for cobalt (Dabeka and McKenzie, 2005 pers. comm.; Health Canada, 2004b; JWEL, 2004a; Dabeka and McKenzie, 1995). The cobalt concentrations reported by these different studies are comparable; however, the databases do not include an analysis of green leafy vegetables.

The datasets selected for use in the Sudbury HHRA were the consecutive years (1993 to 2000) of the CTDS (Dabeka and McKenzie, 2005 pers. comm.; Health Canada 2004b) because they fulfilled all of the selection criteria and were the most appropriate for cobalt. The datasets were combined to increase the Canadian coverage (eight cities) and the statistical robustness of the data. The Canadian TDS results for 1986 to 1988 were not included because cobalt concentrations in approximately half of the samples were not detected. In order to include all important sources of cobalt, the results for green leafy vegetables provided in JWEL (2004a) were also integrated into the database. The other Port Colborne data were not used because 25% of the food samples analyzed were below the detection limit (1.2 ng/g dw; or ~0.96 ng/g ww for vegetables) (JWEL, 2004a). In contrast, <5% of the most recent Total Diet Study samples were below the detection limit (~0.3 ng/g ww) (Dabeka and McKenzie, 2005 pers. comm.).

***Copper***

Canadian market basket data are available for copper (Dabeka and McKenzie, 2005 pers. comm.; Health Canada, 2004b; JWEL, 2004a). There was good agreement among the results for the CTSD (Dabeka and McKenzie, 2005 pers. comm.; Health Canada, 2004b). The Port Colborne results were lower than the other databases but within the same order of magnitude (JWEL, 2004a).

The copper levels for organ meats were significantly higher than the rest of the meat and poultry samples for all three studies. For example, the mean copper concentrations for the meat category with and without the organ meats for three different studies were: 10,911 and 1,342 ng/g in the 2000 CTDS; 3,496 and 1,006 ng/g in the 1993 to 1999 CTDS; and, 21,935 and 685 ng/g in the Port Colborne study (refer to section D.2.1 of Appendix D for further discussion on organ meats).

The databases selected for use in the Sudbury HHRA were the consecutive years (1993 to 2000) of the CTDS (Dabeka and McKenzie, 2005 pers. comm.; Health Canada, 2004b) because they fulfilled all of the selection criteria and were the most appropriate for copper. The datasets were combined to increase the Canadian coverage (eight cities) and the statistical robustness of the data.

***Lead***

There were a number of Canadian datasets available for lead, all conducted as part of the CTDS (Dabeka and McKenzie, 2005 pers. comm.; Health Canada, 2004b; Dabeka and McKenzie, 1995). The databases selected for use in the Sudbury HHRA were Dabeka and McKenzie (2005, pers. comm.) because they fulfilled all of the selection criteria and were found to be the most appropriate for lead.

The CTDS lead results for 1993 through to 1999 (Health Canada, 2004b) could not be used because the accuracy of the data at near-detection limit measurements was poor due to the accidental contamination of the samples (Dabeka and McKenzie, pers. comm. 2005). The older Total Diet Study results were also not used because lead concentrations in environmental media and biological tissues/fluids are generally much lower today than in the 1970s and 1980s (ATSDR, 1999). In addition, older Canadian diet studies (and presumably other studies in which lead was measured in various media) used analytical techniques that may not have been sensitive enough for the prescribed purpose.

***Nickel***

There are two Canadian market basket studies available for nickel (JWEL, 2004a; Dabeka and McKenzie, 1995). Refer to Appendix D (Table D.6) for an overview of these studies, as well as other non-Canadian surveys not used in the HHRA. While food products were analyzed for nickel as part of the CTDS conducted in 2000, the data were accidentally contaminated by nickel-coated skimming (sampling) cones during analyses (Dabeka and McKenzie, 2005 pers. comm.). Therefore, the 2000 CTDS concentration data for nickel was not usable for the current study.

There was good agreement in nickel concentrations between the 1986-1988 Total Diet Study (Dabeka and McKenzie, 1995) and Port Colborne market basket study (JWEL, 2004a) for the categories that were uncooked (*i.e.*, other vegetables; sugars and sweets; fats, nuts and oils; and, beverages). However, the Port Colborne mean nickel concentrations in the cooked food categories were approximately three times lower than those calculated for those reported in the CTDS by Dabeka and McKenzie (1995).

Concern has been expressed (*i.e.*, JWEL, 2004a) with the interpretation of the nickel concentrations in the cooked food analyzed in the 1986 to 1988 Canadian Total Food Study (*i.e.*, Dabeka and McKenzie, 1995). The food samples were prepared using new stainless steel frying and roasting pans. Food was analyzed before and after cooking and the results indicated that significant nickel contamination occurred, particularly by roasting some of the meat samples (Dabeka and McKenzie, 1995). Jacques Whitford (JWEL, 2004a) conducted an extensive literature review and a series of experiments to explore the role of cooking with stainless steel utensils on the leaching of nickel into food samples (some key papers include

Christensen and Moller, 1978; Kuligowski and Halperin, 1992; Kumar *et al*., 1994; Tupholme *et al*., 1993). Their review revealed that significant nickel is leached during cooking; however, this contamination decreases to negligible amounts after the first few uses of the utensil (JWEL, 2004a). They also conducted a screening-level cooking study with a well-used stainless steel frying pan and ceramic pan. This study demonstrated that the foods were not contaminated by nickel during normal preparation and cooking (use of "well used" stainless steel pan) (JWEL, 2004a). Thus, they concluded that contamination of the food items in the Dabeka and McKenzie (1995) study does not appropriately characterize the long term contribution of nickel to the general public from cooking using stainless steel utensils.

The U.S. FDA (2004) also conducted an analysis for nickel in market basket foods. Approximately 320 different food items were sampled for the period 1991 to 2002, from over 36 cities across the United States. The foods were prepared as they would be consumed (table-ready), and three samples per food item were combined to form a single analytical composite for each food item. Details of the nature of the cooking of the samples were not available. Nickel was not detected in 23% of the 6,459 samples evaluated. In the calculation of the mean values for each food item, U.S. FDA (2004) used a value of zero for samples with nickel levels below detection. The results of this study were also lower than the Dabeka and McKenzie (1995) analysis, but higher than the Port Colborne analysis (JWEL, 2004a). There was good agreement between the JWEL and the U.S. FDA dataset for fish and shellfish, dairy products, root vegetables, other vegetables and fats and oils. For cereals and grains the U.S. FDA data was in good agreement with the Dabeka and McKenzie database. All three databases agreed well for other vegetables.

JWEL (2004a) cooked their food samples using ceramic and well-used stainless steel cooking utensils. Based on this review, the Port Colborne data were determined to be the most recent and reliable food dataset for a Canadian population. Therefore, the dataset selected for use in the Sudbury HHRA was market basket data sampled from the Port Colborne area (JWEL, 2004a) because it fulfilled all of the selection criteria and was found to be the most appropriate for nickel. The Port Colborne data were gathered in 2002 with between one and 10 samples per food item (this number also includes replicated and duplicates). The Port Colborne market basket study found 16.5% of food items were below the MDL (0.0091 mg/kg dw) (JWEL, 2004a). Most samples with non-detectable concentrations of nickel were in the meat, poultry, eggs, milk and milk products food categories.

***Selenium***

No Canadian food data for selenium were found in the published literature. A recent survey conducted by the U.S. FDA, which analyzed foods consumed in the United States during the period of 1991 to 2004, detected selenium in 5,586 out of 10,026 food samples (U.S. FDA, 2004). The Canadian Nutrient File (2001) contained data on the selenium content of foods; however, the data were derived from American sources (*i.e.*, United States Department of Agriculture) and were reported in a manner that is inconsistent with the purpose of the Sudbury HHRA (*e.g.*, g/cup; g/8 nuts; g/sandwich). Thus, the FDA (2004) data were selected as the dataset to use in the Sudbury HHRA because of the robustness of the dataset (>10,000 food samples) and the lack of suitable Canadian alternatives. The mean selenium values reported by the U.S. FDA assumed that any non-detectable values were equal to zero. For the purpose of this study, the recalculated UCLs on the mean assumed that non-detectable values were equal to half the detection limit.

Table 4.22 provides a summary of the COC concentrations calculated for the market basket EDI.

**Table 4.22 95% UCLM values for COC concentrations in market basket foods (ng/g)**

| Food Category | Arsenic | Cobalt | Copper | Lead | Nickel | Selenium |
|---|---|---|---|---|---|---|
| Dairy Products | 6.7 | 10.4 | 357 | 6.0 | 15.0 [a] | 71.6 |
| Meat, Poultry and Eggs | 33.6 | 13.6 | 7,260 | 7.2 | 20.6 | 264 |
| Meat, Poultry and Eggs (without organ meats) | 15.2 | 10.8 | 1,060 | 6.6 | 22.4 | 247 |
| Fish and Shellfish | 2,070 | 9.3 | 1,320 | 6.9 [a] | 37.0 | 426 |
| Bakery Goods and Cereals | 28.1 | 24.8 | 1,790 | 12.0 | 165 | 134 |
| Root Vegetables | 10.2 | 32.9 | 1,070 | 7.3 | 75.0 | 13.9 |
| Other Vegetables | 22.1 | 13.4 | 1,240 | 5.0 | 280 | 23.3 |
| Fruit and Fruit Juices | 6.7 | 25.5 | 1,740 | 14.3 | 79.5 | 9.2 |
| Fats and Oils | 26.7 | 22.4 | 251 | 0.4 [a] | 57.0 [a] | 25.3 |
| Nuts and Seeds | 21.4 | 62.9 | 14,000 | 13.5 [a] | 2,000 [a] | 316 |
| Sugar and Candies | 22.6 | 23.8 | 1,400 | 40.5 | 272 | 20.7 |
| Infant Formula | na | 4.6 | 899 | na | 11.0 | 23.0 |

[a] Maximum value was used when the calculated 95% UCLM value was greater than the maximum value in the data set.
na = not available

### 4.1.4 Summary of EPC Data used in the HHRA

Table 4.23 provides a summary of the exposure point concentration (EPC) data outlined in the previous sections and Chapter 3, which were used in the current assessment.

**Table 4.23 Summary of 95% UCLM values for all Exposure Point Concentrations (EPCs) used in the HHRA**

| Community of Interest | As [a] | Co | Cu | Pb | Ni | Se |
|---|---|---|---|---|---|---|
| ***Soil Concentrations*** | | | **µg/g** | | | |
| Coniston | 12 | 19 | 320 | 52 | 433 | 1.3 |
| Copper Cliff | 19 | 33 | 1370 | 98 | 976 | 7.5 |
| Falconbridge | 79 | 57 | 1010 | 82 | 1070 | 3.1 |
| Hanmer | 4.3 | 6.6 | 67 | 19 | 68 | 0.68 |
| Sudbury Centre | 7.2 | 11 | 204 | 36 | 210 | 1.3 |
| Typical Ontario Resident | 17 | 21 | 85 | 43 | 120 | 1.9 |
| ***Dust Concentrations (calculated)[b]*** | | | **µg/g** | | | |
| Coniston | 87 | 98 | 204 | 127 | 221 | 49 |
| Copper Cliff | 98 | 113 | 298 | 150 | 273 | 77 |
| Falconbridge | 142 | 130 | 276 | 143 | 280 | 61 |
| Hanmer | 67 | 74 | 136 | 98 | 137 | 41 |
| Sudbury Centre | 76 | 85 | 182 | 116 | 183 | 49 |
| Typical Ontario Resident | 95 | 101 | 145 | 121 | 158 | 54 |
| ***Air Concentrations (outdoor and indoor)*** | | | **µg/m$^3$** | | | |
| Coniston | 0.0024 | 0.00087 | 0.016 | 0.0080 | 0.012 | 0.0034 |
| Copper Cliff | 0.0050 | 0.0025 | 0.081 | 0.022 | 0.059 | 0.0055 |
| Falconbridge | 0.0024 | 0.0025 | 0.026 | 0.015 | 0.028 | 0.0034 |
| Hanmer | 0.0056 | 0.00066 | 0.099 | 0.0098 | 0.012 | 0.0040 |
| Sudbury Centre | | | | | | |
| *Combined data (2 stations)* | 0.0061 | 0.0097 | 0.17 | 0.025 | 0.095 | 0.0092 |
| *Travers Street only* | 0.0090 | 0.018 | 0.20 | 0.031 | 0.26 | 0.014 |
| Typical Ontario Resident | 0.001 | 0.0019 | 0.0091 | 0.0080 | 0.0014 | 0.0019 |
| ***Drinking Water*** | | | **µg/L** | | | |
| Coniston | 1.1 | 0.2 | 45 | 0.31 | 53 | 1.3 |
| Copper Cliff | 2.5 | 0.05 | 170 | 1.4 | 49 | 3 |
| Falconbridge | 2.6 | 0.2 | 30 | 0.97 | 32 | 2.5 |
| Hanmer | 1.5 | 0.06 | 65 | 0.49 | 0.8 | 1.3 |
| Sudbury Centre | 1.1 | 0.2 | 45 | 0.31 | 53 | 1.3 |
| Typical Ontario Resident | 0.64 | 0.088 | 0.41 | 2.2 | 1.9 | 1.6 |
| ***Home Garden – Below Ground Vegetables*** | | | **µg/g wet weight** | | | |
| Coniston | 0.0069 | 0.024 | 0.81 | 0.26 | 0.56 | 0.029 |
| Copper Cliff | 0.0088 | 0.019 | 1.2 | 0.13 | 1.7 | 0.42 |
| Falconbridge | 0.025 | 0.13 | 1.2 | 0.23 | 3.7 | 0.016 |
| Hanmer | 0.042 | 0.10 | 1.1 | 0.25 | 0.31 | 0.10 |
| Sudbury Centre | 0.0075 | 0.017 | 1.1 | 0.075 | 0.79 | 0.040 |
| ***Home Garden - Above Ground Vegetables*** | | | **µg/g wet weight** | | | |
| Coniston | 0.0069 | 0.21 | 0.54 | 0.095 | 0.57 | 0.030 |
| Copper Cliff | 0.016 | 0.13 | 0.92 | 0.13 | 1.8 | 0.68 |
| Falconbridge | 0.052 | 0.11 | 0.75 | 0.038 | 2.0 | 0.02 |
| Hanmer | 0.0046 | 0.0074 | 0.46 | 0.089 | 0.28 | 0.0083 |
| Sudbury Centre | 0.0067 | 0.027 | 0.75 | 0.094 | 0.75 | 0.059 |

**Table 4.23 Summary of 95% UCLM values for all Exposure Point Concentrations (EPCs) used in the HHRA**

| Community of Interest | As [a] | Co | Cu | Pb | Ni | Se |
|---|---|---|---|---|---|---|
| ***Home Garden – Fruits*** | | | **µg/g wet weight** | | | |
| All COI | 0.0063 | 0.019 | 0.90 | 0.046 | 2.7 | 0.058 |
| ***Wild Berries*** | | | **µg/g wet weight** | | | |
| All COI | 0.0052 | 0.016 | 0.68 | 0.074 | 0.71 | 0.016 |
| ***Local Commercial Produce*** | | | **µg/g wet weight** | | | |
| Root Vegetables | 0.0086 | 0.037 | 1.0 | 0.11 | 0.91 | 0.13 |
| Above Ground Vegetables | 0.0079 | 0.038 | 0.71 | 0.078 | 1.1 | 0.10 |
| Fruit | 0.0061 | 0.035 | 0.65 | 0.042 | 1.5 | 0.024 |
| ***Fish and Wild Game*** | | | **µg/g wet weight** | | | |
| Wild Game | 0.00013 | 0.040 | 0.68 | 0.0040 | 0.62 | 1.4 |
| Fish | 0.00022 | 0.019 | 0.52 | 0.30 | 0.032 | 2.0 |
| **Market Basket Foods - TEDIs** | | | **µg/g** | | | |
| Infant Formula | $7.2 \times 10^{-6}$ | 0.0046 | 0.90 | 0.0023 | 0.011 | 0.020 |
| Dairy | 0.0032 | 0.010 | 0.36 | 0.0060 | 0.015 | 0.072 |
| Meat and Eggs | 0.00046 | 0.011 | 1.1 | 0.0066 | 0.022 | 0.25 |
| Fish | 0.00041 | 0.0093 | 1.3 | 0.0069 | 0.037 | 0.43 |
| Root Vegetables | 0.0043 | 0.033 | 1.1 | 0.0073 | 0.075 | 0.014 |
| Other Vegetables | 0.0093 | 0.013 | 1.2 | 0.0050 | 0.28 | 0.023 |
| Fruits | 0.0022 | 0.025 | 1.7 | 0.014 | 0.080 | 0.0092 |
| Cereals and Grain | 0.0059 | 0.025 | 1.8 | 0.012 | 0.17 | 0.13 |
| Sugar and Sweets | 0.0077 | 0.024 | 1.4 | 0.040 | 0.27 | 0.021 |
| Fats and Oils | 0.0091 | 0.022 | 0.25 | 0.00038 | 0.057 | 0.025 |
| Nuts and Seeds | 0.0073 | 0.063 | 14 | 0.014 | 2.0 | 0.32 |

[a] The arsenic exposure point concentration (see highlighted entries) for all food products (*i.e.*, home garden, local produce, fish and wild game, and market basket foods) were adjusted to represent only the inorganic arsenic fraction content of the food (on which the TRV is based), as follows: all vegetable produce: 0.42, fruits and berries: 0.33, wild game: 0.028, fish: 0.002, infant formula: 0.55 (based upon whole milk), dairy: 0.47, meat and eggs: 0.03, cereals and grains: 0.21, sugars and sweets: 0.34; fats and oils: 0.34, and nuts and seeds: 0.34. Refer to Section 4.1.3 for further discussion of these factor adjustments, and Table 4.22 for the adjustment factors for each specific food grouping.

[b] Indoor dust concentrations calculated based upon regression equation developed from paired soil and indoor dust data collected during the Sudbury indoor dust survey.

### 4.1.5 Exposure Assessment of Carcinogens

As the health endpoint of concern for carcinogenic chemicals in the HHRA framework is considered to be incremental lifetime cancer risk, the exposure period that is assessed is an assumed lifetime (*i.e.,* typically a period of 70 years is assumed; U.S. EPA, 1989). However, for exposure periods that comprise less than 70 years (which is generally the case), the exposures must be amortized (or averaged) over the entire lifetime. Thus, if an individual is exposed to COC for five years, the exposure estimate would typically be multiplied by a factor of 5/70, to yield an amortized exposure estimate. For each exposure scenario assessed, all five receptor age classes (see Chapter 2 of this Volume) were evaluated to provide an evaluation of lifetime cancer risks through the use of a composite receptor.

### 4.1.6 Deterministic v*ersus* Probabilistic Exposure Analysis

Human health risk assessment generally involves assigning numerical values to input parameters in an appropriate exposure or risk model to obtain a quantitative estimate of risk. Numerical values are required for parameters describing contaminant concentrations in environmental media, contaminant fate and transport, human exposure and toxic response. These values may be measured, assumed, prescribed or based on published literature. Variability and uncertainty in the input parameters or risk model result in variability and uncertainty in the estimate of risk. It is important that uncertainty in the model not be confused with variability. Uncertainty derives from a lack of knowledge. Alternatively, variability in the model describes differences in parameter values such as metal concentrations at different locations within the study area, or differences in body weight or food intake rates for individuals (*i.e.*, population heterogeneity).

Traditional deterministic methods of quantitative risk assessment use single, or "point estimate" values for input parameters and produce a single estimate of risk or hazard. While input parameters may be selected with some knowledge of their inherent variability or uncertainty, a deterministic analysis does not normally provide any information on the variability or uncertainty of the resulting risk estimate. For example, although input values are often selected to represent either average or reasonable maximum exposure conditions, the location of the point estimate of risk in the context of its potential range and distribution cannot be determined directly. A discrete, or deterministic, sensitivity analysis may provide some indication of the potential range of estimated risk values, but the variability of, and hence confidence in, the risk estimate remains unknown.

For the current assessment, both "average" (*i.e.,* central tendency estimate, or CTE) and "reasonable maximum exposure" (RME) exposures were evaluated, as recommended in U.S. EPA Superfund guidance (U.S. EPA, 2001b). The former was characterized by the arithmetic mean, while the latter was based upon the 95$^{th}$ upper confidence limit (UCL) on the mean (or the highest measured concentration, if the UCL exceeded the maximum).

The outcome of a deterministic risk assessment model does not provide any information on its underlying distribution, nor does it indicate the likelihood that the risk estimates accurately represent upper percentiles or the central tendency (*e.g.,* the mean, mode, median) of the underlying risk distribution. Consequently, it can be difficult to identify instances where the deterministic risk estimate may be over- or understating the actual potential for risk (aside from basic numeric comparisons between the CTE and RME estimates).

In cases where risks to human health estimated using deterministic methods are clearly not negligible or obviously unacceptable, a probabilistic risk assessment (PRA) may be useful to better characterize risk. PRA uses probability distributions to characterize the inherent variability and uncertainty in input parameters, and produces a probability distribution of estimated exposure or risk. The exposure distribution can be directly compared to a toxicity benchmark to estimate the probability of exceedance. As such, a PRA accounts for natural variability and uncertainty to produce estimated probabilities of exceeding toxicity benchmarks or probabilities of effects of differing magnitude. Evaluating, calculating, and conveying the degree and magnitude of variability and uncertainty in each of the components of the risk assessment process provides decision makers and the public with a strong scientific foundation for understanding risk and evaluating the believability of the final risk estimates.

A deterministic analysis is almost always undertaken as part of a site-specific quantitative human health risk assessment. Its purpose may be one or more of the following: to screen contaminants, exposure pathways and/or receptors; to determine the need for a PRA; to determine the sensitivity of the risk estimate to key assumptions (by discrete sensitivity analysis); and/or to assess the requirement for additional data collection (U.S. EPA, 1999b). In many cases, the risk assessment may not proceed beyond the deterministic step, either because risks were shown to be negligible based upon a deliberately conservative analysis, or were shown to be obviously unacceptable. Sometimes, however, the deterministic analysis serves as a scoping stage for a more detailed probabilistic risk assessment.

Prior to proceeding with a PRA, the risk assessor should consider whether a probabilistic analysis is necessary and/or appropriate, given the objectives of the assessment and the availability of data. A probabilistic analysis necessarily involves a greater commitment of resources to conduct the analysis and to report and present the results. In practice, probabilistic analyses are more commonly conducted with large, complex sites, where the consequences of an incorrect decision are great (*e.g.,* overlooking possibility of catastrophic events, or spending millions of dollars on unnecessary cleanup). In these cases, the additional resources required of a PRA are justified to ensure a complete understanding of risk and ultimately to ensure that a cost-effective risk management strategy can be developed.

Typically, HHRAs determine the worst case point estimate exposure for all receptors and scenarios first. This helps identify areas where more detailed approaches (such as further data collection or the use of probabilistic modelling techniques) should be used to refine assumptions to enable a more site-specific and realistic exposure assessment. However, in situations where the worst case point estimate approaches show no potential risks to receptors, it is typically unnecessary to apply more detailed approaches.

As part of the overall study design of the current HHRA, data were collected to permit both deterministic and probabilistic analysis of risks in the GSA. However, following the detailed peer review conducted by the International Expert Review Panel (IERP), recommendations from the IERP resulted in the elimination or modification of many of the underlying data distributions which would be used in a probabilistic risk assessment. As a result of the removal/adjustment to these distributions, the use of probabilistic risk assessment in the current HHRA no longer provided realistic and useful results, as they would be based on a very small number of PDFs. As a result, risk estimates for the current HHRA have been based upon the results of the deterministic analyses of the reasonable maximum exposure (RME) scenarios (see discussion below).

### 4.1.7 Exposure Estimation Methods

Two general point-estimate exposure estimates, the central tendency exposure (CTE) and the reasonable maximum exposure (RME), were evaluated for each community of interest (COI), human receptor and COC. For the CTE estimate, human receptor characteristics were defined in such a way as to reflect the central tendency within a given population. Most receptor characteristics (*e.g.,* soil, water and food ingestion rates, *etc.*) were obtained using the 50$^{th}$ percentile values of the sample distribution. The arithmetic mean was selected to represent parameters such as body-weight. Body weight is typically located in the denominator of most exposure calculations and therefore use of the 50$^{th}$ percentile *versus* the arithmetic mean (in the case of a lognormal distribution) would result in an inflated exposure estimate relative to the arithmetic mean body weight. The RME estimate typically employed the use of upper percentiles (typically 90 to 95$^{th}$) for most receptor characteristics, with the exception of food intake rates. As recommended by the IERP, to avoid unrealistic daily caloric intake diets (*i.e.*, multiple 95$^{th}$ percentile ingestions of various food groups), food intake rates were based upon mean or median values.

As previously discussed, human health risks were calculated for individuals living in five COI within the Greater Sudbury Area (GSA). For comparative purposes, a "Typical Ontario Resident" (TOR) was also evaluated. Individuals were assumed to move in a random fashion within each COI and, over time, come into contact with the exposure point concentration (EPC) of the COC in a variety of environmental media. The EPC for any given environmental media (*e.g.,* air, soil, water, food, *etc.*) was defined as the 95% UCLM for that particular COI.

The following methods summary has been organized by exposure pathway. A discussion of the general assumptions, references and intake rates used within each exposure pathway are provided. Refer to the Problem Formulation Section (Chapter 2) of this volume for receptor- and scenario-specific input

parameters. A comprehensive discussion and description of the equations and algorithms used in the model to estimate exposures can be found in Appendix B.

#### 4.1.7.1 Outdoor/Indoor Air Exposure

The direct air inhalation pathway utilized the basic exposure equations from U.S. EPA (2004a). The 95% UCL on the arithmetic mean air concentrations for each COI were generated using the ProUCL program developed by the U.S. EPA. Based upon discussions with the Technical Committee, it was agreed that indoor air concentrations would be assumed equal to those measured outdoors.

#### 4.1.7.2 Outdoor Soil/Indoor Dust Exposure

***Dermal Contact with Indoor and Outdoor Soil and Dust***

A detailed literature review was conducted regarding the methods used to predict chemical exposures *via* dermal contact with impacted soils and dusts. A number of recent U.S. EPA references were investigated including U.S. EPA (1997a; 2002b; 2004a), Richardson (1997), Burmaster (1998), and Garlock *et al.* (1999). The overall approach used to evaluate exposures *via* direct dermal contact with soil and dust were taken from U.S. EPA (2004a). U.S. EPA (2004a) provided three different methods to assess dermal exposures. The fraction of total surface area method was selected for use in the current assessment.

Indoor and outdoor area-weighted soil adherence values were derived using data presented in U.S. EPA (1997a; 2002b; 2004a). Area-weighted adherence factors were derived using the percentage of the total surface area of each body part (hands, arms, legs and feet) in conjunction with body-part specific adherence values for a given activity. Indoor adherence/loading factors were developed based on children playing indoors on carpeted areas. Adherence factors for adults were selected from Table 6-12 of the U.S. EPA (1997a) and were based on indoor Tae Kwon Doe Activities.

With the exception of the adult receptors, outdoor adherence/loading factors provided by the U.S. EPA (2002b) were used. The "Soccer No.1 activity" study from Table 8-8 of the U.S. EPA (2002b) was selected as a representative activity to correlate adherence/loading factors. Adult adherence/loading factors were selected from Table 6-11 of the U.S. EPA (1997a) in conjunction with the "Groundskeeper No.4" scenario as a representative activity.

For children, teenagers and adults, the percentage of the total surface area for each body part was selected from Tables 6-5 and 6-8 of the U.S. EPA (1997a). The mean percentage of total body surface area reported by U.S. EPA (2002b) was used for infants and preschool children. Indoor (body-part specific)

dust adherence factors for infants, preschool children, children and teenagers from Table 8-8 of the U.S. EPA (2002b) were used in the current assessment.

The U.S. EPA (2002b) presents various clothing scenarios in which 10 to 25% percent of skin surface area is estimated to be exposed. The default value for children is 25% of the $50^{th}$ and $95^{th}$ percentiles of total surface area. It is suggested that estimates of exposed skin could be refined based on seasonal conditions. A prorated, seasonally-adjusted estimate of the area of exposed skin was developed by dividing the year into spring (61 days), summer (92 days), fall (91 days) and winter (121 days) with each season associated with a different fraction of exposed skin. During the spring and fall seasons it was assumed that 15% of the total body surface area would be exposed, while during the summer months, exposure of 25% of the total body surface area was considered to be reasonable. The fraction of skin exposed during the winter season was considered to be much less at only 5% of the total body surface area. A seasonally-adjusted fraction of exposed skin was estimated to be 14.2% based on the duration of each season and the fraction of exposed skin within each season. The ability to come into direct contact with surface soil during the winter season was considered less likely due the additional clothing worn during these months and the fact that much of the ground is either frozen and/or covered with snow.

Issues regarding the correlation between body weight and surface area during Monte Carlo simulations were addressed. An equation developed by Burmaster (1998) was used to relate body weight (BW) and total surface area (SA) as follows:

$$SA\ (m^2) = 0.1025 * BW^{0.6821}$$

The SA/BW ratio approach was developed in order to express surface area and body weight as a direct correlation (*i.e.*, by a factor of 1.0). This approach has been recommended for use by the U.S. EPA (2002b).

Rather than using the whole body surface area data presented by Richardson (1997), the Burmaster equation (above) was used, along with probability distribution functions (PDFs) for body weight from Richardson (1997), to estimate a total body surface area for each receptor type. A 10,000 iteration test conducted using Crystal Ball 7.01 (*i.e.*, to develop a probabilistic distribution of possible body surface area values based upon body weight) provided results consistent with the surface area values reported by Richardson (1997).

The potential for direct contact with surface soil during the winter season was considered less likely due the additional clothing worn during these months and the fact that much of the ground is either frozen and/or covered with snow. A winter covering factor of 10% was applied to the outdoor soil ingestion pathways during the winter season only.

***Incidental Soil and Dust Ingestion***

A significant amount of both regulatory and scientific literature regarding the application of incidental soil intake rates of children for use during chronic exposure assessments was reviewed, including Calabrese *et al.* (1997a;b), U.S. EPA (1997b; 1999c; 2002b; 2004a), Stanek *et al.* (2000; 2001a; 2001b), and Health Canada (2004a).

The U.S. EPA (1997a; 2002b) recommends a mean soil intake rate of 100 mg/day and an upper conservative mean of 200 mg/day. It is unclear whether these values include dust; however, based on the description provided, it appears that they represent soil intake rates only. It is noted that the recommendation of 100 mg/day is highly uncertain and based on a number of different short-term intake studies. It should be noted that the Ontario MOE also recommends the use of 100 mg/day (B. Birmingham, personal communication, 2007). Health Canada (2004a) recommends a soil ingestion rate of 80 mg/day for children and 20 mg/day for all other receptors. Based upon this review and its recommended use by a relevant Canadian regulatory agency, this value was selected for use in the current assessment. However, due to the uncertainty surrounding the selection of this variable, evaluations of potential risk using either soil intake rate was completed as part of the sensitivity analyses (see Chapters 5 and 7 for further discussion).

An issue that has been recognized within the scientific literature is that soil intake rates of children are generally based on short-term (*i.e.,* two to five day) tracer studies. As a result, intake rates developed from these studies may not be able to capture the long-term, day-to-day variation in soil ingestion rates and therefore, may exaggerate long-term average daily intakes.

With the exception of Calabrese *et al.* (1997a,b), intake rates for indoor dust were not identified in the literature. It is noted that several references reported "soil and dust intake rates" combined; however, only Calabrese *et al.* (1997a,b) reported distinct indoor dust intake rates. Given that the Calabrese *et al.* (1997a,b) distribution was associated with negative dust intake rates at the $50^{th}$ percentile, it was decided that the dust intake rate distribution could not be used.

The U.S. EPA's Integrated Exposure Uptake Biokinetic (IEUBK) model for lead in children employs central tendency "total soil and dust" ingestion rates for five individual age classes of children ranging from 85 mg/day to 135 mg/day. These are central tendency values and do not represent the prevalence of *pica* (intentional ingestion of soil) behaviour (refer to Section 6.5 of this volume for further discussion of "pica" children). The IEUBK model also uses a default 45/55 split which assumes that 55% of the total soil and dust ingestion rate is applied to dust while 45% of the intake rate is applied to soil. The relative proportions used as defaults (45% outdoor soil and 55% indoor dust) are discussed in the U.S. EPA's IEUBK guidance as follows: "*The ratio of soil intake to dust intake is not simply proportional to the ratio of the number of waking hours that the child spends outdoors versus indoors. Children spend only 15 to 30% of their waking hours playing outside but are more likely to be in contact with bare soil areas, in locations with large amounts of accessible loose particles, and are likely to wash their hands less often than when they are indoors. The default 45/55 ratio in the model represents our best judgment of a properly weighted ratio for this parameter.*" (U.S. EPA, 1994).

As a result, information provided by the U.S. EPA's IEUBK model was employed to develop indoor dust ingestion rates for preschool children and children alike. As previously discussed, the IEUBK model employs central tendency total soil and dust ingestion rates for five individual age classes of children ranging from 85 to 135 mg/day. The IEUBK model uses a default 45/55 split which applies 55% of the total soil and dust ingestion rate to indoor dust with the remaining 45% being applied to soil.

#### 4.1.7.3 Exposure *via* Home Garden Produce and Wild Berry Consumption

***Consumption of Home Garden Produce***

Each exposure scenario assumed that individuals living within any one of the five COI may consume produce grown locally (from anywhere within the GSA) and/or from their own backyard gardens. As a result, fruit and vegetable concentrations reported in the 2003 Vegetable Garden Survey (data report provided in Appendix E) were first organized by vegetable type (*i.e.,* above-ground produce, below-ground root vegetables, and fruits) and origin (*i.e.,* within a designated COI or outside of a COI). All garden vegetable samples collected within the GSA were used to characterize the metal concentrations in various types of local produce. Vegetable samples taken from within one of the five defined COI were used to characterize metal concentrations in COI-specific home garden produce. The 95% UCL on the arithmetic sample mean was used to define EPCs for produce.

***Consumption of Wild Blueberries***

The gathering and consumption of local wild blueberries is common practice in the GSA and, therefore, was considered as a separate exposure pathway. Data from the 2003 Vegetable Garden Survey (refer to Appendix E) provided metal concentrations in wild blueberry samples (n=10) collected within the GSA. The Local Food Consumption Survey (complete technical report found in Appendix K) reported mean and median blueberry consumption rates of 173 and 12 cups/yr, respectively. The median intake rate of 12 cups/yr year was used to form the central tendency estimate of the consumption rate of local wild blueberries. The reported mean intake rate of 173 cups/yr is equivalent to approximately 40 kg of blueberries per person per year. This consumption rate was considered high compared to the upper 95$^{th}$ percentile consumer-only consumption rate of "other berries" of 1.28 g/kg/day (or 33 kg/year) (U.S. EPA, 1997b). The "other berries" food category includes all berries other than strawberries, including a wide range of commercially frozen and canned berry produces (*e.g.,* pie fillings, cranberry sauces, blackberries juices, *etc*.).

For the purpose of the point estimate assessment, the CTE estimate employed a daily wild blackberry consumption rate equivalent to the reported median intake of 12 cups/yr (or approximately 0.12g/kg/day). For the RME estimate, it was assumed that an individual may consume up to twice the amount of blueberries as the CTE estimate or 0.24 g/kg/day (approximately 5.5 kg/yr for a female adult). For the stochastic (or probabilistic) assessment, a continuous triangular PDF was employed utilizing consumption rates of zero for the non-consumer, 0.12 g/kg/day for the consumer at the central tendency, and 0.24 g/kg/day as a high end consumer.

#### 4.1.7.4 Background Market Food Basket Exposure

Market basket exposures were defined as exposures resulting from the consumption of typical supermarket foods. Metal concentrations in market basket foods were considered representative of the typical levels observed in supermarket foods across Canada. Refer to Appendix D for the complete Market Basket Estimated Daily Intakes Report. For the current assessment, market basket exposures were classified as background exposures (*i.e.,* exposures which are independent of the GSA).

The 95% UCLM intake rates of specific food groups were used to determine market basket exposures for both the CTE and RME estimates. Food intake rates provided by Richardson (1997) were based on a 24-hour recall study collected during the 1972 to 1973 National Food Consumption Survey (NFCS). The lognormal probability distributions representing food consumption provided by Richardson (1997) do not reflect long-term food consumption patterns of an individual, but rather the variability of reported

consumption rates of many individuals over a 24-hour recall period. It is not considered realistic (nor is it recommended) to use the Richardson (1997) probability density functions (describing variability in 24-hour consumption rates) when characterizing long-term exposures.

When conducting point estimate assessments (*i.e.,* the CTE and RME estimates), the application of successive upper percentile food consumption rates (*e.g.,* 95$^{th}$ percentiles) would suggest that an individual might consume every food group at the 5$^{th}$ highest intake rate (observed during NFCS 24-hour recall study) for an entire year, or in the case of a carcinogenic assessment, a lifetime. As this approach is not reasonable, the 95% UCLM food group-specific intake rates were used for both the CTE and RME estimates. Raw food intake data from the NFCS was provided by Richardson, 2005 pers. comm. The statistical software package ProUCL (U.S. EPA, 2004b) was used to generate stable 95% UCLM estimates on these data.

#### 4.1.7.5 Exposure *via* Drinking Water Ingestion

Exposure point concentrations (EPCs) in drinking water were defined for each COI using the 95% UCLM. Receptor-specific water intake rates and body weights were used to estimate chemical-specific daily intake rates for all individuals (*e.g.*, preschool children, children, teenagers and adults) and COI. The CTE estimate employed the 50$^{th}$ percentile water intake rates provided by Richardson (1997). The RME exposure estimate employed the 95% UCLM drinking water intake rates. Each exposure estimate (*i.e.,* the CTE and RME) used age-specific mean body weights provided by Richardson (1997).

#### 4.1.7.6 Exposure *via* Ingestion of Local Food

Local foods were defined as those food stuffs which were either grown or caught within the GSA including garden vegetables, fruits, wild blueberries, fish and wild game. The proportion of an individual's total daily intake that is comprised of food items originating from within the GSA is highly uncertain. However, information provided by the Local Food Consumption Survey (see Appendix K) and the U.S. EPA (1997b) were used to approximate the proportion of an individual's daily food intake that may be of local origin. In addition to defining an individual's consumption of local foods (*i.e.,* foods grown or caught within the GSA), the proportion of locally derived foods which may originate from an individual's home garden was also explored. This further division of home grown produce *versus* local foods was considered necessary since metal content in local foods may differ between different COI.

The total daily food intake of an individual was kept constant by expressing local food intake rates as a proportion of the total daily intake for a specific food group. In other words, it was not assumed that an

individual consuming local food was consuming more total food per day than an individual not consuming local products, but rather, it was assumed that an individual would derive a certain proportion of their total food intake from local sources.

***Local Fruits and Vegetables***

Information gathered from the Local Food Consumption Survey (see Appendix K) and Volume II of the U.S. EPA Exposure Factors Handbook (U.S. EPA, 1997b) was used to approximate the amount of fruits and vegetables an individual might consume from the GSA and/or from an individual home garden. The U.S. EPA (1997b) used the Nationwide Food Consumption Survey (NFCS) data to generate intake rate approximations of different home produced foods. At the time of the publication, the latest NFCS had been conducted from 1987 to 1988. The sample size of the NFCS in 1987 was approximately 4,300 households or 10,000 individuals.

The U.S. EPA (1997b) cautions that consumption rate data are based on short-term observations (*i.e.,* seven days) and therefore are not appropriate for use in long-term exposure assessments. This is particularly true for home produced vegetables and fruits since consumption rates would be highly correlated to season (*i.e.,* spring, summer, fall and winter). As a result, the U.S. EPA (1997b) attempted to derive a long-term distribution of the average daily intake rates of home produced foods from the short-term data available for major food groups (vegetables, fruits and meats). The approach attempted to account for variability in consumption rates from one season to the next. According to U.S. EPA (1997b), the seasonally adjusted distributions for a given region (*e.g.,* the north eastern region) were derived by averaging the intake rates for each of the four seasons (spring, summer, winter and fall).

The seasonally adjusted percentiles representing consumer-only (*i.e.*, excluding all individuals who did not consume that particular food item from statistical analyses) consumption rates of home produced vegetables in the Northeast region (which includes Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island and Vermont) were compared to the site-specific intake rate data collect for the Local Food Consumption Survey (see Appendix K).

The Local Food Consumption Survey did not provide information with regard to the proportion of foods that come from an individual's garden but rather the amount of local foods consumed from within the GSA. Local foods were defined as foods being grown or caught from somewhere within the GSA. For produce, the Local Food Consumption Survey provided intake rates (in cups per year) for root vegetables

(including potatoes, carrot and green onions), above-ground vegetables (including cucumbers, lettuce, bean and tomatoes) and fruit (strawberries and wild blueberries).

The units of measure reported in the Local Food Consumption Survey made comparative analysis with other data sets difficult, if not impractical, since intake rate data from the U.S. EPA (1997b) are typically expressed as a weight of food (g) per unit of body weight (kg) per day (*i.e.*, g/kg/day). To compare the site-specific survey data with information provided by the U.S. EPA, the consumption rates provided in the Local Food Consumption Survey were converted from a "cups/person/year" estimate to a "g/kg/day" approximation assuming a vegetable density of 1 g/cm$^3$ (*i.e.*, one cup ~ 237 cm$^3$ = ~ 237 g), a constant intake rate throughout the year, and an adult body weight of 63 kg. Although the conversion may not be entirely accurate, it allowed for a general comparison between site-specific survey data and home produced seasonally adjusted (local produce eaters only) intake rates provide by the U.S. EPA (1997b).

The intake rate data provided in the Local Food Consumption Survey were also highly skewed (not unexpected for such a limited recall-based survey). Reported mean intake rates were often more than twice the reported median value, indicating a highly skewed distribution. Reported median intake rates for total vegetables from the GSA were five times higher than the reported 50$^{th}$ percentile seasonally adjusted (eaters only) intake rate of home produced vegetables.

Given the uncertainty in the unit conversion from cups/yr to g/kg/day, the degree of skewness observed, and the fact that survey data were based on a single recall event, it was decided that data from the U.S. EPA (1997b) would be used to help approximate local vegetable intake. The fraction of home produced foods, as reported by U.S. EPA, 1997b (Table 13-70), were used in combination with Canadian food intake rates from the National Food Consumption Survey (NFCS). To derive local vegetable consumption rates for the CTE estimate, the fraction of home produced root and exposed vegetables for the Northeast region of the U.S. of 0.018 and 0.062 were multiplied by the central tendency intake rates derived from the NFCS for root and leafy vegetables, respectively. For a female adult receptor, central tendency intakes of local root and leafy vegetables of 0.038 g/kg/day and 0.096 g/kg/day were generated, respectively. The RME estimate employed the reported fractions of root and exposed vegetables of 0.106 and 0.233, respectively, that are home produced for those individuals who garden. This resulted in a local root and leafy vegetable intake rate for the female adult of 0.30 g/kg/day and 0.5 g/kg/day, respectively. The total RME local vegetable intake rate of 0.8 g/kg/day for a female adult corresponds to approximately the 68$^{th}$ percentile of the U.S. EPA (1997b) seasonally adjusted consumer only home-grown intake rate of total vegetables in the Northeast region of the United States. Correcting for the percentage of individuals

consuming home-grown produce (*i.e.,* 16.5%), 0.8 g/kg/day corresponds to 96th percentile per capita home-grown intake rate of total vegetables for northeast region.

For locally grown fruits, the site-specific food intake survey indicated that cultivated strawberries were the main source of locally derived fruit (wild blueberries were considered separately). The median intake for cultivated strawberries originating from the GSA was reported to be 10 cups/yr, equivalent to approximately 0.1 g/kg/day or approximately 2.3 kg of local strawberries per person per year. This intake rate was used to derive a fraction of fruit coming from the GSA. A female adult consumption rate for fruit and fruit juices of 3.1 g/kg/day was used to derive a CTE fraction for local fruit of approximately 0.03 (or 3%). The RME local fruit consumption rate for the female adult was 0.22 g/kg/day, assuming a RME fraction for local fruit of 5.3%.

***Local Fish***

The consumption of local fish was considered for two distinct populations including the general population of the GSA and an angler sub-population within the GSA. The population of anglers would be expected to consume much larger quantities of local fish than the typical GSA resident. The Local Food Consumption Survey provided self-reported consumer only intake rates of anglers for the top four most commonly consumed fish species (walleye, trout, pike and perch). The survey also reported the number of times per year individuals (from within both the general and angling populations) would consume locally caught fish. These data were available for the most commonly consumed fish species only.

Walleye, trout, pike and perch were the species most commonly consumed by the general GSA population. Daily consumption rates of freshwater fish were estimated using the consumption frequency data provided by the Local Food Consumption Survey and information from the Great Lakes Sport Fish Consumption Advisory Task Force (GLSFATF, 1993). The GLSFATF (1993) suggests that a typical serving of fish is approximately 227 grams. Combining the site-specific consumption frequency data with an assumed serving size of 227 grams produced a mean (or CTE) intake rate of fish for the general population of 12.44 g/day. See Table 4.24 for an overview of the local fish consumption rates.

One standard deviation from the reported mean consumption frequency was used to approximate the RME consumption rate of 33 g/day. The CTE and RME fish intake rates for the sub-population of anglers were determined to be 35.6 and 85.4 g/day, respectively. These data are elevated relative to the recommended U.S. EPA (1997b) mean and upper 95$^{th}$ percentile freshwater fish intake rates for recreational anglers of eight and 25 g/day, respectively. For the general population, the U.S. EPA

(1997b) provides a long-term mean consumption rate of approximately 6.6 g/day of freshwater fish which is recommended for use in long-term exposure assessments.

**Table 4.24 Local Fish Consumption Rates**

| Fish Species | General Sudbury Population | | Angling Population | |
|---|---|---|---|---|
| | CTE (meals/yr) | RME (meals/yr)[a] | CTE (meals/yr) | RME (meals/yr)[a] |
| Walleye | 5.4 | 12.23 | 18.2 | 42.2 |
| Trout | 4.7 | 10.71 | - | - |
| Pike | 5.9 | 21.33 | 17.6 | 46.6 |
| Perch | 4 | 13.52 | 21.5 | 48.5 |
| Sum (meals/yr) | 20.00 | 57.79 | 57.30 | 137.30 |
| TOTAL (g/day)[b] | 12.44 | 35.94 | 35.64 | 85.39 |

[a] RME intake rates were derived by adding one standard deviation to the reported mean intake frequency data. As a result, RME frequency data reflect the mean plus one standard deviation.

[b] The number of meals/yr was converted to g/day by assuming a serving size 227g (8 ounces) and a constant intake rate of fish over the entire year.

For the current assessment, the CTE and RME estimates (for the general population) employed local fish consumption rates equivalent to 12.44 g/day and 35.94 g/day, respectively. For the angling population, CTE and RME estimates employed intake rates of 35.64 and 85.39 g/day. It should be noted that these intake rates are significantly greater than the 95$^{th}$ percentile intake rate for fresh water anglers of 25 g/day reported by the U.S. EPA (1997b).

***Local Wild Game***

Wild game tissue concentrations predicted for the ERA were used in the HHRA in combination with consumption rates provided by the Local Food Consumption Survey to determine exposures to COC from the consumption of local wild game. As a conservative measure, predicted wild game concentrations from "Zone 2" (as defined in Volume III of the Sudbury Soils Study Report) were used since metal concentrations from within Zone 2 were the highest among all other zones or COI. A comparison between those ecological receptors considered as potential wild game (i.e., deer, moose, mallard duck and grouse) indicated that moose tissue had the highest metal concentrations. As a result, COC concentrations in moose meat, predicted from within Zone 2, were used as EPCs for wild game. A detailed discussion of the methodology used in the ERA to derive the predicted wild game concentrations can be found in Section 4.1.1.8.

According to the Local Food Consumption Survey, local hunters and anglers reported consuming five types of game caught within the GSA, including grouse, moose, deer, wild rabbit and ducks/geese. Grouse, moose and deer were identified as the three most commonly consumed game, by 65, 62 and 48%

of hunters, respectively. Less than 20% of all hunters interviewed indicated consuming wild rabbit while less than 5% of the general Sudbury population sampled indicated consuming rabbit. The Local Food Consumption Survey provided self-reported intake rates for specific sub-populations (*i.e.,* Whitefish First Nation and anglers and hunters). Self-reported intake rates for the general Sudbury population were not available.

The method used to approximate local wild game intake on a g/day or g/kg/day basis was the same method used to approximate local fish intake rates. For the general Sudbury population, CTE and RME wild game consumption rates of 7.2 and 13.0 g/day were estimated, respectively. CTE and RME wild game intake rates for those individuals who reported hunting were approximately 29.8 and 60.3 g/day, respectively. Again, these estimates were derived using an assumed 227 g serving size and the reported mean and upper percentile consumption frequencies provided by the Local Food Consumption Survey.

For comparative purposes, Table 13-44 of the U.S. EPA (1997b) provides consumer only game consumption rate percentiles for those individuals who identified themselves as hunters. Approximately 12% of hunters reported consuming wild game at a mean and 95$^{th}$ percentile consumer only intake rate of 1.04 and 2.9 g/kg/day (or approximately 81.12 and 226.2 g/day), respectively.

**Table 4.25 Local Wild Game Consumption Rates**

| Species | General Sudbury Population | | Hunting Population | |
|---|---|---|---|---|
| | CTE (meals/yr) | RME (meals/yr)[a] | CTE (meals/yr) | RME (meals/yr)[a] |
| Moose | 4.5 | 6.74 | 24.6 | 48.6 |
| Deer | 4.3 | 8.46 | 10.8 | 20.8 |
| Grouse | 2.8 | 5.73 | 12.5 | 27.5 |
| Sum (meals/yr) | 11.60 | 20.93 | 47.90 | 96.90 |
| TOTAL (g/day)[b] | 7.21 | 13.02 | 29.79 | 60.26 |

[a] RME intake rates were derived by adding one standard deviation to the reported mean intake frequency data. As a result RME frequency data reflect the mean plus one standard deviation.

[b] The number of meals/yr was converted to g/day by assuming a serving size 227g (8 ounces) and a constant intake rate of fish over the entire year (GLSFATF, 1993).

For the general GSA population, CTE and RME wild game consumption rates of 7.21 and 13.02 g/day were selected (see Table 4.25). For the sub-population of hunters within the GSA, CTE and RME wild game consumption rates of 29.79 and 60.26 g/day were employed.

### 4.1.8 Development of the Risk Assessment Modeling Tool

To appropriately evaluate potential exposures to each of the COC, it is important to utilize exposure estimation methodologies, which incorporate the most up-to-date information and techniques for estimating exposure and risk.

Exposure estimation in the current HHRA was facilitated through the use of an integrated multi-pathway environmental risk assessment model. The model is spreadsheet based (MS Excel). Models of this type have been used in hundreds of peer-reviewed human health risk assessments, including those conducted for contaminated sites, smelters, refineries, incinerators, landfills and a variety of other industrial facilities. The current version of this model incorporated the latest techniques and procedures for exposure modelling developed by various regulatory agencies (*e.g.,* U.S. EPA, MOE, CCME, Cal/EPA, U.S. EPA Region VI, WHO, *etc*.) and published academic and scientific literature sources. The model integrated recent statistical and probabilistic techniques, and was capable of conducting complex modelling involving human receptors, and a myriad of exposure pathways.

To ensure transparency in the HHRA, and to facilitate any future Ministry and/or peer reviews of the HHRA, all assumptions, equations, and parameters used in the assessment, as well as sample calculations, are provided in Appendix B.

## 4.2 Hazard Assessment

The objectives of the hazard assessment (also termed *toxicity assessment*) are to:

- Provide the reader with an understanding of the toxicological effects that have been reported to be associated with exposure to the COC by various routes;
- Identify whether each COC is considered to cause carcinogenic (non-threshold) or non-carcinogenic (threshold) effects; and,
- Identify the most appropriate and scientifically-defensible exposure limits against which exposures can be compared to provide estimates of potential health risks.

*Toxicity* refers to the potential for a chemical to produce any type of damage, permanent or temporary, to the structure or functioning of any part of the body. The toxicity of a chemical depends on the amount of chemical taken into the body (referred to as the "dose") and the duration of exposure (*i.e.,* the length of time the person is exposed to the chemical). For every chemical, there is a specific dose and duration of exposure necessary to produce a toxic effect in humans (this is referred to as the "dose-response relationship" of a chemical). The toxic potency of a chemical (*i.e.,* its ability to produce any type of damage to the structure or function of any part of the body), is dependent on the inherent properties of the chemical itself (*i.e.,* its ability to cause a biochemical or physiological response at the site of action), as well as the ability of the chemical to be absorbed into the body (*i.e.,* bioavailability), and then to reach the site of action. The dose-response principle is central to the human health risk assessment methodology.

There are two main types of dose-response relationships for chemicals:

**Threshold Response Effects**: For some chemicals, it is thought that there is a dose-response threshold below which no adverse effects would be expected to occur. This relationship is true for all chemicals that do not cause cancer by altering genetic material (*e.g.,* most metals). Thresholds are generally assumed for non-carcinogens because, for these types of effects, it is generally believed that homeostatic, compensating, and adaptive mechanisms must be overcome before toxicity is manifested. Exposure limits derived for threshold-response chemicals are called reference doses (RfD), acceptable daily intakes (ADI), tolerable daily intakes (TDI) or permissible daily intakes (PDI) and are generally derived by regulatory agencies such as Health Canada and the U.S. Environmental Protection Agency (U.S. EPA). These values indicate doses of chemicals that individuals can receive on a daily basis without the occurrence of adverse health effects. Exposure limits derived for

threshold-response chemicals are typically expressed as :g/kg body weight/day, and are typically based on experimentally-determined "No-Observed-Adverse-Effect Levels" (NOAELs), with the application of extrapolation factors that are often referred to as "safety factors" or "uncertainty factors" (U.S. FDA, 1982; U.S. EPA, 1989; Health Canada, 1993). The magnitude of these factors is dependent on the level of confidence in the available toxicology database, and reflects differences in species, duration of exposure, sensitivity, and overall quality of available data (*i.e.,* the weight-of-evidence of the supporting data).

**Non-threshold Response Effects**: For these chemicals, it is assumed that there is no dose-response threshold. This means that any exposure greater than zero is assumed to have a non-zero probability of causing some type of response or damage. This relationship is typically used for chemicals which can cause cancer by damaging genetic material. Under a "no threshold" assumption, any exposure has some potential to cause damage, so it is necessary to define an "acceptable" level of risk associated with these types of exposures. For the purposes of evaluating exposures to chemicals in the environment, the "acceptable" level of risk is usually defined as a risk of one-in-one hundred thousand to one-in-one million. These numbers can be better explained as the daily dose that may cause an additional incidence of cancer (*i.e.,* one cancer that would not be expected in the absence of the exposure) in a population of one hundred thousand (or a million) people exposed every day over their entire lifetime. The acceptable level of risk is a policy rather than a scientific decision, and is set by regulatory agencies, as opposed to risk assessors. For example, the MOE has indicated that an incremental lifetime cancer risk level less than one-in-one million would be considered a *de minimis* risk level; in other words, a risk which is considered so small, it is of little or no significance and is acceptable from a regulatory perspective (MOEE, 1987). Exposure limits derived for non-threshold chemicals that are believed to be potential carcinogens are typically expressed as "increased risk per unit of dose". These potency estimates are called cancer slope factors (SF) or cancer potency factors (*e.g.,* $[\mu g/kg \text{ body weight/day}]^{-1}$). These values are derived using a mathematical model-unit risk estimation approach with the built-in assumption that the condition of "zero increased risk of cancer" would only be observed when the dose is zero.

It must be recognized that the assumption of no dose-response threshold for carcinogens is an assumption which is not directly testable by experimentation. Thresholds may exist, even for assumed non-threshold chemicals and effects. The "no threshold" assumption ignores a large number of factors, such as the ability of the body to repair damage to genetic material, that are known to be important responses of people to naturally-occurring genotoxic carcinogens. Exposure to small concentrations of chemicals

which have the potential to cause cancer happens on a daily basis to everyone in the world, because non-threshold chemicals (along with other chemicals which do not cause cancer) are present in soils, air, food and water, either from natural sources or as a result of human activities. The human body has many ways of handling these substances once they enter the body. In many cases, the body can repair damage that may be caused by exposures to low levels of carcinogenic chemicals; therefore, adverse effects do not necessarily occur.

The development of toxicological criteria or exposure limits for any given chemical must consider factors which affect the potential toxicity of that chemical. These factors may be scenario-specific, such as variation in duration or levels of exposure. Where possible, it is important that exposure limits be derived from "realistic" exposure situations that are representative of those occurring under the conditions assessed in the HHRA. For many chemicals, the toxic endpoint is also dependent on the route of exposure, as exposure *via* different routes may impact different tissues, such as those at the site of entry. In such a case, different exposure limits may be identified or developed for the different routes of exposure. Toxic potency may be modified by species- or individual-specific factors such as the ability to resist, repair or adapt to the effects of chemical exposures. In these situations, separate exposure limits might be used to ensure protection of sensitive sub-populations.

Exposure limits for chemicals are based on scientific information, professional judgement and technical review by experienced scientists with expertise in a wide range of scientific disciplines. Exposure limits are derived based on the most sensitive endpoints in individuals (*e.g.,* cancer, organ damage, neurological effects, reproductive effects, *etc.*). In many cases, large uncertainty factors (*i.e.,* 100-fold or greater) are used in establishing exposure limits for chemical causing effects that are expected to have thresholds. Thus, exceedance of the exposure limit does not necessarily mean that adverse effects will occur. Rather, this result would necessitate a more detailed evaluation of both exposure and the toxicity-based exposure limit to better understand the likelihood of adverse effects occurring. Exposure rates less than an exposure limit are usually considered unlikely to be associated with adverse health effects and are, therefore, less likely to be of concern. As the frequency or magnitude of exposures exceeding the exposure limit increase, the probability of adverse health effects in a human population is usually presumed to increase, subject to scientific judgement and critical evaluation of the exposure limit and the exposure estimate, as discussed above. However, it should not be categorically concluded that all exposures below an exposure limit will be unlikely to result in adverse health effects or that all exposures above such a limit are likely to result in adverse health effects.

### 4.2.1 Overview of Exposure Limits Selected for the HHRA

A detailed toxicological assessment was conducted for each COC, involving identification of mechanism of action and relevant toxic endpoints, and determination of receptor- and route-specific toxicological criteria (see Appendix A). These profiles were not intended to provide comprehensive reviews of the available toxicological and epidemiological literature on the various COC. Rather, the purpose of the toxicological profiles was to: i) summarize the most relevant toxicological and epidemiological information on the substances; ii) outline any recent information that may challenge previous findings; and iii), provide supporting rationale for the exposure limits selected for use in the human health risk assessment of the Sudbury area. The toxicological reviews are based primarily on secondary sources, such as ATSDR toxicological profiles and other detailed regulatory agency reviews, and are supplemented with recent scientific literature. For all profiles the primary literature was searched from the date of last major review to the present. Thus, for lead, for instance, primary literature from 1999 to 2005 was considered as the latest ATSDR summary at the time the profile was completed was dated 1999. The review of primary literature was mainly to determine if any recent information exists that may challenge previous findings.

While the specific requirement of the MOE is that all toxicological criteria used in a human health risk assessment assume values that are recommended by regulatory agencies such as Health Canada, the U.S. EPA, and the MOE itself, a comprehensive review of the critical toxicological literature was conducted in order to put the predicted risks associated with COC into perspective. For those COC where toxicological criteria have been developed by a regulatory agency, the development of these values considered sensitive subgroups of the population, both through use of the most stringent scientific data, as well as application of uncertainty factors in the derivation of the criteria. This yielded final toxicological criteria that are considered protective of the individuals most sensitive to the toxicity of the chemical, whether due to differences in genetics, life stage, nutrition, or health status.

A thorough review of the scientific and regulatory literature pertaining to the toxicity of many of the COC was previously conducted by the MOE (MOE, 2002) and Jacques Whitford (JWEL, 2004b) as part of recent HHRA work related to the Rodney Street Community in Port Colborne. Exposure limits for the COC in the current HHRA have been identified from regulatory agencies such as MOE, Health Canada, U.S. EPA, U.S. Agency for Toxic Substances and Disease Registry (ATSDR), California Environmental Protection Agency Office of Environmental Health Hazard Assessment (CalEPA OEHHA), U.S. Centers for Disease Control (CDC), the European Union (EU), and the World Health Organization (WHO).

### 4.2.2 Selection of Toxicological Criteria for the HHRA

This section provides an overview of the regulatory exposure limits considered for use in the current assessment. MOE guidance discourages the development of *de novo* toxicological criteria (exposure limits) when health based expousre limits are available from major health agencies. The exposure limits (or toxicological criteria) employed in the current assessment were obtained from a review of toxicological criteria from various regulatory agencies including the MOE, Health Canada, the Canadian Council of Ministers of the Environment (CCME), the WHO, California Environmental Protection Agency Office of Environmental Health Hazard Assessment, U.S. Agency for Toxic Substances and Disease Registry, and Centers for Disease Control and the U.S. EPA. The toxicological criteria used in this assessment reflect the approach preferred by the MOE, which requires the use of toxicity assessments published by reputable regulatory agencies such as those mentioned above. Review of the regulatory exposure limits (toxicological criteria) was supplemented by detailed toxicological assessments conducted for each COC, involving identification of mechanism of action and relevant toxic endpoints, and determination of receptor- and route-specific toxicological criteria. Together, this information was used to select toxicological criteria for each COC that are based on the best available science. In some instances, several regulatory agencies and/or authorities have recommended different exposure limit values for the same chemical. In this situation a rationale has been provided for the use of one regulatory criterion over another for use in this study.

The U.S. EPA derives exposure limits for both threshold and non-threshold effects when data are available. The RfD and RfC are based on the assumption that a threshold exists for certain toxic non-carcinogenic effects. In general, the RfD (or RfC) is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (U.S. EPA, 2005).

For a number of chemicals, exposure limits are not always available for all exposure routes of concern. In these circumstances, exposure limits may be extrapolated from other routes. For example, it is common in human health risk assessments to assess the risks posed by dermal absorption of a chemical based on the exposure limit established for oral exposure (U.S. EPA, 1989; 1992). The systemic dose absorbed dermally is scaled to the "equivalent" oral dose by correcting for the bioavailability of the dermally-applied chemical relative to an orally-administered dose.

The relative absorption difference between the oral and dermal routes of exposure can be expressed as a relative absorption factor ($RAF_{dermal}$). This factor, calculated as follows, is applied to dermal exposure estimates to adjust these exposures prior to comparison with oral exposure limits when route-to-route extrapolation is necessary.

$$RAF_{dermal} = \frac{AF_{dermal}}{AF_{oral}} x\,100$$

Where:

| | | |
|---|---|---|
| $RAF_{dermal}$ | = | relative absorption factor for dermal exposure (%). |
| $AF_{dermal}$ | = | the fraction of the applied chemical absorbed through the skin. |
| $AF_{oral}$ | = | the fraction of the ingested chemical absorbed into the bloodstream. |

It must be recognized however that route extrapolation is only appropriate where effects are systemic in nature, and not closely associated with the point of exposure.

Certain COC, specifically cobalt and nickel, are known immuno-sensitizers (or dermal sensitizers), and can result in skin irritation (*i.e.*, contact dermatitis) to sensitive individuals under certain circumstances. However, there is little available data for use in establishing an exposure limit which is protective of skin sensitization following dermal exposure. For a further discussion of this topic, refer to Section 6.7 of this volume.

Table 4.26 summarizes the toxicological criteria selected for use in the Sudbury HHRA. Refer to Appendix A for detailed toxicological reviews of each COC in the HHRA.

**Table 4.26 Summary of Toxicological Criteria chosen for the Sudbury Human Health Risk Assessment**

| Chemical | Route | Toxicological Criterion [a] | | Endpoint | Study | Regulatory Agency |
|---|---|---|---|---|---|---|
| Arsenic | Oral | RfD | 0.3 µg/kg/day | Hyperpigmentation, keratosis, possible vascular complications (human) | Tseng *et al.*, 1968; Tseng, 1977 | U.S. EPA, 1993 |
| | | $SF_o$ | 0.0015 $(\mu g/kg/day)^{-1}$ | Skin cancer, basal and squamous cell carcinoma (human) | Tseng *et al.*, 1968; Tseng, 1977 | U.S. EPA, 1998 |
| | Inhalation | Chronic REL | 0.03 $\mu g/m^3$ | Decreased fetal weight; increased incidences of intrauterine growth retardation and skeletal malformations in mice | Nagymajtényi *et al.*, 1985 | OEHHA, 2000 |
| | | $SF_i$ (IUR) | 0.015 $(\mu g/kg/day)^{-1}$ [$4.3x10^{-3}$ $(\mu g/m^3)^{-1}$] | Lung cancer (human) | Enterline and Marsh, 1982; Higgins, 1982; Brown and Chu, 1983a,b,c; Lee-Feldstein, 1983 | U.S. EPA, 1998 |
| Cobalt | Oral | RfD | 10 µg/kg/day | Polycythemia | Davis and Fields (1958) | ATSDR (2001) |
| | Inhalation | RfC | 0.5 $\mu g/m^3$ | Interstitial lung disease | Sprince et al., 1988 | RIVM (Baar *et al*, 2001) |
| Copper | Oral | UL | 140 µg/kg/day | Liver damage (human) | Pratt *et al.*, 1985 | IOM, 2001; Health Canada, 2005 |
| | Inhalation | TCA | 1 $\mu g/m^3$ | Subchronic NOAEC (respiratory and immunological effects) (rabbits) | Johansson *et al.*, 1984 | RIVM (Baars *et al.*, 2001) |
| Lead | Oral, Inhalation, Dermal | $IOC_{POP}$ | 1.85 µg/kg/day | Subclinical neurobehavioural and developmental effects (child) | Various | MOE, 1996a; MOE, 1994 |
| Nickel | Oral | RfD | 20 µg/kg/day | Decreased body and organ weight (rats) | Ambrose *et al.*, 1976 | U.S. EPA, 1996 |
| | Inhalation | RfC | 0.02 $\mu g/m^3$ (total nickel) | Respiratory effects (lung inflammation and lung fibrosis) | European Commission DG Environment, 2001 | OJEU, 2005 |
| Selenium | Oral | RfD/TRV | 5.00 µg/kg/day | Selenosis, including hair loss and nail sloughing (human)<br><br>Clinical selenosis | Yang and Zhou, 1994<br><br>Yang *et al.*, 1989a,b | IOM, 2000; Health Canada, 2005<br><br>U.S. EPA, 1991a |
| | Inhalation | Chronic REL RfC | 20 $\mu g/m^3$ | Hepatic, cardiovascular, neurological effects (human) | Yang *et al.*, 1989a,b Dudley and Miller, 1941 | OEHHA, 2001 |

[a] RfD = reference dose; SFo = oral slope factor; SFi = inhalation slope factor; IUR = inhalation unit risk; REL = reference exposure level; TCA = tolerable concentration in air; UL = upper intake level; $IOC_{POP}$ = intake of concern (population) – unlike an RfD or RfC (or similar benchmark), there is no established threshold or 'acceptable' or 'safe' levels for critical health effects of lead, at or below which no adverse health effects would be expected to occur.; TRV = toxicity reference value.

Note: For chemicals with no identified inhalation toxicological criteria, it was assumed that inhalation bioavailability and toxic potency is equivalent to that which occurs *via* the oral exposure route.

***Inhalation Conversion Factors***

As of January, 1991, IRIS and NCEA databases no longer present RfDs or SFs for the inhalation route (U.S. EPA, 2004c. These criteria have been replaced with reference concentrations (RfC) for noncarcinogenic effects and unit risk factors (URF) for carcinogenic effects. However, for purposes of estimating risk and calculating risk-based concentrations, inhalation reference doses (RfDi) and inhalation slope factors (SFi) are preferred. This is not a problem for most chemicals because the inhalation toxicity criteria are easily converted. To calculate an RfDi from an RfC, the following equation and assumptions may be used for most chemicals:

$$RfD_i = RfC\,(mg/m^3)\,x\frac{20(m^3/day)}{70(kg)}$$

Likewise, to calculate a SFi from an inhalation URF, the following equation and assumptions may be used:

$$SF_i = URF\,(mg/m^3)^{-1}\,x\frac{70(kg)}{20(m^3/day)}$$

### 4.2.3 Summary of Toxicological Profiles

### *Inorganic Arsenic*

Essentiality

Arsenic has not been demonstrated to be essential in humans (WHO-IPCS, 2001).

Exposure Limits

The following paragraphs relate to inorganic arsenic species only, as all regulatory TRVs that exist for arsenic have been developed from data on inorganic arsenic exposure. Furthermore, it is inorganic species of arsenic that human receptors are most likely to come into contact with in the GSA.

The following organizations were consulted to select exposure limits for arsenic: the U.S. EPA; MOE; ATSDR; Health Canada; the Dutch National Institute for Public Health and the Environment (RIVM); NRC; WHO; and, OEHHA.

While some recent studies suggest that certain organic arsenicals (such as pentavalent MMA and DMA) may be of similar or greater toxic potency than inorganic arsenic species (See Appendix A1 for details), a

recent review of the toxicokinetics and toxicology of methylated arsenicals by Cohen *et al.*, (2006) notes that the animal carcinogenicity data for $MMA^V$ and $DMA^V$ are equivocal. These authors also state that the metabolism and disposition of $MMA^V$ and $DMA^V$, when formed endogenously during the metabolism of inorganic arsenic, differs from the metabolism and disposition of these methylated species when exposure is exogenous. Furthermore, the trivalent arsenicals that known to be cytotoxic and indirectly genotoxic *in vitro* are formed in negligible amounts in organisms exposed exogenously (ingestion) to $MMA^V$ or $DMA^V$ due to low cellular uptake and limited metabolism of these compounds. Cohen *et al*., (2006) conclude that at anticipated environmental exposures to $MMA^V$ and $DMA^V$, carcinogenic risk to humans is unlikely. In a science issue paper produced by the U.S. EPA Office of Pesticide Programs on a mode of carcinogenic action for $DMA^V$ (cacodylic acid) (U.S. EPA OPP, 2005), it is also noted that there are differences in methylation efficiency and cellular uptake between direct exposure to $DMA^V$ and exposure to inorganic arsenic, with subsequent metabolism to $DMA^V$ and other methylated species. The U.S. EPA paper also notes that direct exposure to $DMA^V$ results in the production of fewer arsenical metabolites relative to metabolism that occurs following direct exposure to inorganic arsenic. Thus, exposure to inorganic arsenic results in a more complex mixture of metabolites and transformation products. The U.S. EPA paper also states that there is presently insufficient evidence to establish pentavalent MMA and DMA species as the ultimate carcinogenic forms of inorganic arsenic. Rather, it is likely that several inorganic and organic arsenical species may be involved in various modes of action in different target tissues. For $DMA^V$, U.S. EPA OPP (2005) suggests that this substance is a threshold carcinogen with a carcinogenic mode of action that is non-linear. As such, a reference dose has been proposed using benchmark dose modelling.

Exposure limits derived by the U.S. EPA were selected for use in this assessment, with the exception of the inhalation RfC, for which the U.S. EPA has not derived a value. Thus, the chronic REL developed by OEHHA was used as a threshold inhalation exposure limit.

Risk assessment experience in several Ontario communities (*e.g.,* Deloro, Wawa, Port Colborne) has revealed that arsenic is a complex substance to evaluate in human health risk assessments. It is important to evaluate arsenic exposures and risks using a weight-of-evidence approach that includes risk assessment, biomonitoring (urinary arsenic), predictive modelling and medical surveillance to collectively and definitively address concerns related to arsenic exposures at contaminated sites (Sigal *et al,* 2002a; Sigal *et al,* 2002b). This approach has been successfully applied in other communities in Ontario where arsenic has been a concern (*e.g.,* Deloro, Wawa and Port Colborne) and has proved effective in ensuring public safety and satisfying the concerns of the local community and regulators.

The cancer potency of arsenic continues to be a source of controversy in the risk assessment and management of arsenic-contaminated sites. The use of U.S. EPA slope factors to estimate possible cancer risks to people through all pathways (air, water, food, soils) consistently results in risk values from natural (*i.e.,* background) sources at or higher than *de minimis* risk levels. Several studies and reviews have questioned the relevance of the Tiawanese dataset for the North American population (U.S. EPA, 2007; Lamm et. al., 2004; Brown and Chen, 1995). For example, Lamm et. al. (2004) considered the relationship between arsenic exposure through drinking water and bladder cancer mortality. County specific mortality ratios were considered for 133 counties across the U.S. where the primary source of drinking water was groundwater. No arsenic-related increase in bladder cancer mortality was found over an exposure range of 3 to 60 µg/L. In Ontario, background arsenic soil levels (17 µg/g) and the generic residential/parkland soil criterion (25 µg/g) are associated with predicted incremental lifetime cancer risk levels in the one-in-one-hundred thousand range. Cancer risk estimates well above the *de minimis* risk level are also routinely predicted for arsenic exposures associated with typical North American diets, air quality and regulated North American drinking water supplies. These elevated arsenic risk levels that result from typical and/or natural exposure conditions create challenges in communicating risk estimates for both incremental and total arsenic exposures. In discussing arsenic risk estimates in a HHRA, it is critical to provide additional perspective using information from a weight-of-evidence approach that includes a variety of "tools" in addition to risk assessment, such as bio-monitoring, predictive modelling and medical surveillance. In combination, these tools can be helpful for regulators and other stakeholders in considering the real-world implications of hypothetical risk predictions based upon HHRA.

### *Oral Exposure Limits*

Non-Carcinogenic (Threshold) Effects

The U.S. EPA (1993) calculated an oral RfD of 0.3 µg As/kg body weight/day based on the epidemiological studies of chronic exposure to arsenic through drinking water (Tseng *et al.,* 1968; Tseng, 1977). Critical effects were hyperpigmentation, keratosis, and possible vascular complications at a lowest-observable-adverse-effects-level of 14 µg As/kg body weight/day. The RfD was based on a NOAEL of 0.8 µg As/kg body weight/day, with the application of an uncertainty factor of three to account for both lack of data on reproductive toxicity in humans, and for differences in individual sensitivity. The U.S. EPA (1993) noted some limitations of the studies, in that the exposure levels were not well-characterized (particularly from foods) and other contaminants were present. Also, there was not a clear consensus among U.S. EPA scientists on the oral RfD, and arguments were made for alternate

values that are within a factor of two or three of the currently recommended RfD value (*i.e.*, 0.1 to 0.8 µg/kg/day) (U.S. EPA, 1993). New data that could possibly impact on the recommended RfD for arsenic will be evaluated by the U.S. EPA Work Group as it becomes available. Confidence in the chosen principal study and the resulting oral RfD is considered medium. MOE (1996b) adopted 0.3 µg As/kg body weight/day, based on information provided on IRIS in 1993, as the chronic reference dose as part of the Guideline for Use at Contaminated Sites in Ontario. This conservative exposure limit was in use by the U.S. EPA (1998) and the value of 0.3 µg As/kg body weight/day was selected as the oral exposure limit for non-carcinogenic effects in this study.

Carcinogenic (Non-threshold) Effects

Arsenic exposure *via* the oral route was considered by the U.S. EPA to be carcinogenic to humans, based on the incidence of skin cancers in epidemiological studies examining human exposure through drinking water in Taiwan (Tseng *et al.,* 1968; Tseng, 1977). Based on the application of a linear-quadratic mathematical model to the data from these studies, the U.S. EPA (1998) calculated an oral slope factor of 0.0015 (µg As/kg body weight/day)$^{-1}$. The slope factor (SF) is based on the assumption that carcinogenic effects do not have a threshold (*i.e.,* dose-response relationship is linear to zero exposure). It was assumed that the Taiwanese individuals had a constant exposure from birth. It was also assumed that males consumed 3.5 L drinking water per day, and females consume 2.0 L per day. Doses were converted to equivalent doses for U.S. males and females based on differences in body weights and differences in water consumption and it was assumed that background skin cancer risk in the U.S. population would be similar to the Taiwanese population. The multistage model with time was used to predict dose-specific and age-specific skin cancer prevalence rates associated with ingestion of inorganic arsenic; both linear and quadratic model fitting of the data were conducted.

Recently, there has been concern on the part of regulators regarding the applicability of the arsenic cancer potency estimates for cancers at other sites (specifically bladder cancer) in setting exposure limits for arsenic. The National Research Council (NRC) (1999; 2001) recently re-evaluated drinking water criteria for the United States, based on bladder cancer incidence data in the Taiwanese population as presented in Wu *et al.* (1989), Chen *et al.* (1992) and Smith *et al.* (1992). NRC (1999; 2001) emphasized that the evaluation of cancer potency factors for bladder cancer has been limited by the amount and the quality of data available for use in the linear model. While the bladder cancer value would yield a greater cancer potency than that based on skin cancer, these data are still limited by many of the same problems as the potency factor for skin cancer, including large uncertainty of total daily exposure to inorganic arsenic

(*i.e.,* the poor linkage between water concentrations of arsenic and individual exposure, and lack of data on arsenic intake from food), concomitant exposures to other chemicals and carcinogens (which would be especially important if arsenic is a cancer promoter), and differences in nutritional and health status between Taiwanese and North American populations. As the intended use of the cancer potency factor is in the estimation of risk to a particular population in comparison to a "background" or "typical" population, and risks for both will be assessed with the same methodologies and the same exposure limit, the use of the skin cancer potency factor is considered acceptable and conservative.

MOE (1996b) selected 0.00175 (µg As/kg body weight/day)$^{-1}$ as the oral cancer potency factor as part of the *Guideline for Use at Contaminated Sites in Ontario*, based on information provided on IRIS in 1993. This number was considered outdated and was not used in the current study.

The SF of 0.0015 (µg As/kg body weight/day)$^{-1}$, corresponding to an RsD of 0.00067 µg As/kg body weight/day for an acceptable risk level of one-in-one million, was adopted as the oral exposure limit for carcinogenic effects of arsenic for this assessment.

***Inhalation Exposure Limits***

Non-cancer (Threshold) Effects

The U.S. EPA has not established an inhalation reference concentration or dose for arsenic. Thus, the chronic REL developed by OEHHA (2000) was used.

The OEHHA (2000) used the study by Nagymajtenyi *et al.*, (1985) as the basis for deriving the chronic REL. This was accomplished by using the average experimental exposure for the LOAEL group (determined to be 33 µg As/m$^3$) and applying a cumulative uncertainty factor of 1,000 (10-fold each for use of a LOAEL, interspecies extrapolation and intraspecies differences in sensitivity) to yield a chronic REL of 0.03 µg As/m$^3$. According to the OEHHA (2000), route-to-route conversion of the LOAEL in the key study indicates that this chronic REL should also be protective of non-cancer adverse effects that have been observed in studies with oral exposures, either in food or drinking water. Also, OEHHA considers that had available human data been used instead of animal data in the REL derivation, a similar value would have been obtained. Thus, the chronic REL from animal data is believed to be protective of potential adverse health effects in humans.

Cancer (Non-threshold) Effects

The U.S. EPA (1998) considers arsenic to be a non-threshold carcinogen. Based on this assumption, the U.S. EPA (1998) calculated an inhalation unit risk value of 0.0043 (µg As/$m^3$)$^{-1}$, based on studies by Brown and Chu (1983a,b,c), Lee-Feldstein (1983), Higgins (1982), and Enterline and Marsh (1982) which indicated increased lung cancer mortality of exposed populations. A geometric mean was obtained for data sets obtained with distinct exposed populations (Anaconda smelter and ASARCO smelter), and then the final estimate was the geometric mean of those two values. It was assumed that the increase in age-specific mortality rate of lung cancer was a function only of cumulative exposures. The unit risk was converted to a slope factor of 0.015 (µg As/kg body weight/day)$^{-1}$ assuming a 70 kg adult breathes 20 $m^3$/day. It should be noted that all of the studies used to derive the U.S. EPA unit risk value had a number of confounding factors and uncertainties. These included: confounding by concurrent exposure to airborne dusts, sulphur dioxide and other chemicals; lack of measured air concentrations in some studies; failure to consider latent periods for lung cancer development; and, confounding by smoking.

***Dermal Exposure Limit***

There are currently no dermal arsenic exposure limits that have been developed by regulatory agencies. Route-to-route extrapolation was used to derive an appropriate limit for the current assessment.

## *Cobalt*

Essentiality

Cobalt is an essential micronutrient in humans and most other organisms, as it is a required element in vitamin B12, and is also associated with the regulation of several cofactors and enzymes, and the production of erythropoeitin (Lison, 1996). The Recommended Dietary Allowance (RDA) for vitamin B12 is 2.4 µg/day for adults, which corresponds to 0.1 µg/day of cobalt (ATSDR, 2001). Due to its essentiality, cobalt occurs in many tissues of individuals with no known occupational or environmental exposure, with the highest concentrations occurring in the liver, where vitamin B12 is stored (ATSDR, 2001). Adverse health effects will typically occur only at doses that exceed the daily nutritional requirements for cobalt.

Exposure Limits

ATSDR, U.S. EPA, MOE and RIVM were the regulatory agencies consulted to select exposure limits for cobalt.

For the current assessment, cobalt has not been considered a non-threshold carcinogen by the inhalation or oral routes of exposure. There is inadequate information available from oral studies to determine whether or not cobalt is carcinogenic *via* this route. IARC classifies cobalt compounds as "possibly carcinogenic to humans" and ACGIH classifies cobalt in category A3 - confirmed animal carcinogen with unknown relevance to humans. Furthermore, under the old 1986 Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1986), cobalt is classified as group B1 (Probable Human Carcinogen), based on limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals, as evidenced by increased incidence of alveolar/bronchiolar tumors in both sexes of rats and mice (U.S. EPA, 2002a). Under the U.S. EPA (1999b) cancer guidelines, cobalt is considered likely to be carcinogenic to humans (U.S. EPA, 2002a). Health Canada currently has no TRVs for cobalt and has not classified cobalt compounds as to their carcinogenicity.

***Oral Exposure Limits***

Non-Carcinogenic (Threshold) Effects

The most sensitive indicators of the effects of cobalt following oral exposure appear to be the related to an increase of hemoglobin in both humans and animals and the elicitation of dermatitis in sensitized individuals (ATSDR, 2001). The U.S. EPA (2002a) has reported an oral RfD for cobalt of 20 µg/kg/day. The exposure limit is based upon a study conducted by Duckham and Lee (1976) which demonstrated an increased level of hemoglobin in anemic patients treated therapeutically at a level of 0.18 mg/kg/d. The oral RfD was calculated by dividing this LOAEL by 10 (three to account for the use of a LOAEL, three for deficiencies in the database, primarily the use of a sub-chronic study; uncertainly factor rounded to 10). In the derivation of the *Guideline for Use at Contaminated Sites in Ontario* (MOE, 1996b), MOE utilized an oral RfD of 60 µg/kg/day for cobalt.

ATSDR (2001) derived an oral intermediate-duration MRL of 10 ug cobalt/kg/day. The MRL is based on a LOAEL of 1 mg cobalt/kg/day for polycythemia as reported in a study by Davis and Fields (1958). These authors exposed six male volunteers to 120 or 150 mg/day of cobalt chloride (~1 mg Co/kg/day) for up to 22 days. Exposure resulted in the development of polycythemia in all six patients, with 16 to 20% increases in red blood cell numbers above pre-treatment levels. Oral MRL values were not derived

by ATSDR for acute or chronic exposure to cobalt. An acute MRL was not derived because the reported effects in animals were serious and occurred at levels above those reported in the few available human oral studies. No chronic oral studies were available for humans or animals.

RIVM (Baars et al., 2001) derived a tolerable daily intake (TDI) of 1.4 µg/kg-day based on a LOAEL of 0.04 mg/kg-day for cardiomyopathy in humans after intermediate oral exposure (Morin et al., 1971). RIVM used an uncertainty factor of 30 (three for intra-human variation and 10 for extrapolation to a NOAEL) to yield the TDI.

For the purposes of the current assessment, the ATSDR (2001) MRL (10 µg cobalt/kg/day) has been selected the oral exposure limit for non-carcinogenic effects for the current study. While dated, these study results are consistent with those observed in more recent studies such as Duckham and Lee (1976).

Carcinogenic (Non-threshold) Effects

Cobalt does not appear to cause cancer in humans *via* inhalation, oral, or dermal exposure routes (ATSDR, 2001). No studies were located in the literature reviewed regarding carcinogenic effects in animals after oral or dermal exposure to cobalt. In a recent review of genotoxicity and carcinogenicity studies published between 1991 and 2001, Lison *et al*. (2001) concluded there was no evidence for genotoxic or carcinogenic activity of cobalt in humans. However, a recent study by Hengstler *et al*. (2003) reports that co-exposure to cadmium, cobalt and lead may cause genotoxic effects even at concentrations below current regulatory limits, and that the cancer hazard of cobalt exposure may be underestimated, especially when individuals are co-exposed to cadmium or lead. This hypothesis has not yet been substantiated by other studies identified in the scientific literature.

***Inhalation Exposure Limits***

Non-cancer (Threshold) Effects

The U.S. EPA (2002) have reported an inhalation RfD for cobalt of $5.7 \times 10^{-6}$ mg/kg/d based on an RfC of $2.0 \times 10^{-5}$ mg/m$^3$. The exposure limit is based upon an epidemiological study which showed a NOAEL of 0.0053 mg Co/m$^3$ and a LOAEL of 0.015 mg Co/m$^3$ for decreases in forced vital capacity (FVC), forced expiratory volume in one second (FEV1), forced expiratory flow between 25 and 75% of the FVC (MMEF), and mean peak expiratory flow rate (PEF) in diamond polishers (Nemery *et al.,* 1992). The RfC of $2.0 \times 10^{-5}$ mg/m$^3$ was derived by adjusting the NOAEL of 0.0053 mg/m$^3$ for intermittent exposure (8 hours/24 hours x 5 days/7 days), and dividing by an uncertainty factor of 100 (3 to account for exposure duration that may have been subchronic in some workers, 3 for a lack of inhalation

developmental toxicity studies and a multi-generation reproduction study, and 10 for human variability). While these factors yield a cumulative uncertainty factor of 90, the U.S. EPA rounded up to 100 in this case. ATSDR (2001) also used the Nemery *et al.* (1992) study to develop its inhalation MRL, but only applied a 10-fold safety factor to the time-adjusted NOAEL, resulting in the derivation of a less conservative limit of $1x10^{-4}$ mg cobalt/$m^3$. WHO (2006) have also established a tolerable concentration for inhaled cobalt of of $1x10^{-4}$ mg/$m^3$, and is based on a NOAEL of 0.0053 mg cobalt/$m^3$ in diamond polishers (Nemery *et al.,* 1992).

The Dutch Institute for Public Health (RIVM) (Baars et al., 2001) derived a tolerable concentration in air (TCA) of 0.0005 mg/$m^3$, based on a LOAEL of 0.05 mg/$m^3$ for interstitial lung disease in humans (Sprince et al., 1988). An uncertainty factor of 100 (10 for extrapolation from a LOAEL and a factor of 10 for intrahuman variability) was applied to the LOAEL to yield the TCA. Medium reliability is suggested for this TCA by RIVM (Baar et al, 2001). This exposure limit was selected for use in the current assessment.

Cancer (Non-threshold) Effects

Overall, the weight-of-evidence indicates that cobalt does not cause cancer in humans by the inhalation, oral, or dermal exposure routes. The U.S. EPA (2002a) have classified cobalt as a group B1 Probable Human Carcinogen based on limited evidence of carcinogenicity in humans and sufficient evidence in animals following inhalation exposure. The U.S. EPA (2002a) have derived an inhalation unit risk for cobalt of $2.8x10^{-3}$ (µg Co/$m^3$) based on tumourigenic effects (alveolar and bronchiolar) in rats and mice (Bucher *et al*., 1999; NTP, 1998) which equates to a inhalation slope factor of 9.8 $(mg/kg/d)^{-1}$. No other identified regulatory agencies have derived exposure limits for cobalt based on carcinogenic endpoints. There appears to be a consistent increased risk of respiratory tract cancer in workers co-exposed to both cobalt and tungsten carbide *(i.e.,* "hard metal" workers). However, the exposure conditions experienced by "hard metal" workers would not be expected to occur in the ambient environment.

***Dermal Exposure Limit***

No regulatory dermal exposure limits for cobalt were identified in the literature reviewed for the current assessment. Route-to-route extrapolation was used to derive an appropriate limit for the current assessment.

**Uncertainties in Selected Cobalt Exposure Limits**

The key areas of uncertainty regarding the cobalt toxicity values are summarized below (U.S. EPA, 2002a). It is also important to recognize that the areas of uncertainty noted below apply equally to all available regulatory exposure limits for cobalt compounds.

- While there is evidence of allergic responses in cobalt-sensitized workers available data provide no information on the dose-response relationship of cobalt sensitization, nor is a NOAEL for the elicitation of the allergic response in humans defined by the available studies.
- There is some evidence documenting interrelationships between cobalt and nickel sensitization, such that people sensitized by nickel may have an allergic reaction following cobalt exposure. However, information on this endpoint is not sufficient to quantify.
- U.S. EPA (2002a) notes that confidence in the critical study for the oral RfD is low-to-medium, as it examined a small number of subjects over a subchronic rather than chronic duration. However, it is believed that a sensitive endpoint in a group of sensitive humans was considered in the RfD derivation. The U.S. EPA also notes that confidence in the supporting database is medium, as there supporting studies in both anemic and normal humans, and in animals. However, there are no chronic oral data available and only limited data exists on developmental effects.
- No studies exist that investigated developmental effects after inhalation exposure to cobalt.
- No oral or inhalation exposure multi-generation reproduction studies were located.
- Confidence in the key study that the RfC was derived from is low. Reasons include the fact that this study used a cross-sectional design that investigated only respiratory endpoints, the control group was studied more than one year after the exposed population was studied, one study group was exposed to iron and diamond dust in addition to cobalt (and possibly to asbestos in the past), there was no discussion of duration of exposure. Confidence in the supporting database is medium, as the critical endpoint is well supported by other studies in both humans and animals.
- The precise mechanism of action for cobalt carcinogenicity has not been determined, although a number of potential mechanisms have been identified, with the most likely mechanism being cobalt-induced oxidative stress.

- While available human studies are suggestive of a possible association between cobalt and respiratory tumors, these studies have a number of limitations such as small sample size, inadequate exposure assessment, concurrent exposure to other chemicals, which makes them inappropriate for assessing the carcinogenic potential of cobalt.
- There are no oral studies that investigated the carcinogenic potential of cobalt.
- Available genotoxicity and mutagenicity studies are limited and equivocal with respect to supporting the carcinogenicity of cobalt.

### *Copper*

Essentiality

Copper is an essential trace element that is naturally present in all environmental media (air, water, soil, sediments), as well as all biota and all foods consumed by humans. The primary source of copper in humans is the diet. It is estimated that the typical daily copper intake from food is around 1.0 to 1.3 mg/day for adults (ATSDR, 2002). The World Health Organization (WHO, 1998) reports that total daily intake of copper in adult's ranges between 0.9 and 2.2 mg, with most studies indicating daily copper intakes at the lower end of this range. WHO (1998) notes that intakes may occasionally exceed 5 mg/day. In some cases, drinking water may also make a substantial additional contribution to the total daily copper intake, particularly if corrosive waters remain in copper pipes for prolonged periods. Other common environmental routes of exposure, such as inhalation and dermal uptake, are insignificant relative to oral consumption of dietary items. For example, inhalation adds approximately 0.0003 to 0.002 mg/day from dusts and smoke (WHO, 1998). Dermal absorption contributes even less to total daily copper intake.

Among the essential roles of copper in the body are incorporation into at least 30 metalloenzymes involved in such biochemical processes as hemoglobin formation, iron metabolism, carbohydrate metabolism, catecholamine biosynthesis, cellular respiration, free radical defenses, neurotransmitter function, connective tissue biosynthesis (cross-linking of collagen, elastin, and hair keratin) (ATSDR, 2002; WHO, 1998). In a number of these enzymes, copper is an essential co-factor required for enzyme function, while in others, copper confers an appropriate structure for catalytic activity. No other elements are known to be able to substitute for copper in these enzymes (WHO, 1998). Copper is also essential in the maturation of neutrophils (Percival, 1995), and plays an important role in the regulation of gene transcription (Dameron *et al.,* 1991; Zhou and Theil, 1991; Gralla *et al.,* 1991; Carry *et al.,* 1991; Jungmann *et al.,* 1993).

As copper is an essential element, its uptake, metabolism and excretion are physiologically regulated, and most tissues of the body have measurable amounts of copper associated with them. It has been estimated that the whole human body contains 100 to 150 mg copper at any given time (WHO, 1998). All mammals have metabolic mechanisms that maintain copper homeostasis (a balance between metabolic requirements for copper and prevention against accumulation to toxic levels, such that copper levels are generally maintained within a range that avoids both deficiency and excess). Copper homeostasis involves physiological regulation of absorption, cellular uptake, intracellular transport, sequestration/storage, cellular efflux, and excretion from the body (ATSDR, 2002). Table 4.27 presents a summary of human nutritional requirements for copper.

**Table 4.27 Nutritional Requirements for Copper**

| Agency | Infants | Children | Adults |
|---|---|---|---|
| U.S. Food and Nutrition Board, Institute of Medicine, National Academy of Science (2000)[a] | Adequate Intake<br>0 to 6 mths: 200 µg/day<br>7 to 12 mths: 220 µg/day | Estimated Average Requirement<br>1 to 3 yrs: 260 µg/day<br>4 to 8 yrs: 340 µg/day<br>9 to 13 yrs: 540 µg/day<br>14 to 18 yrs: 685 µg/day<br>14 to18 yrs (pregnancy): 785 µg/day<br><br>Recommended Dietary Allowance[b]<br>1 to 3 yrs: 240 µg/day<br>4 to 8 yrs: 440 µg/day<br>9 to 13 yrs: 700 µg/day<br>14 to 18 yrs: 890 µg/day<br>14 to 18 yrs (pregnancy): 1,000 µg/day | Estimated Average Requirement<br>19 to >70 yrs: 700 µg/day<br>19 to 50 yrs (pregnant): 800 µg/day<br><br>Recommended Dietary Allowance[b]<br>19 to >70 yrs: 900 µg/day<br>19 to 50 yrs (pregnant): 1,000 µg/day |

[a] Values are also currently recommended by Health Canada (2004b) as Dietary Reference Intakes.
[b] Recommended Dietary Allowance is the average daily dietary nutrient intake level sufficient to meet the nutrient requirement of 97 to 98% of healthy individuals in the gender and age group for which it was developed.

As shown in Table 4.27, the recommended copper intake values are within the range of estimated daily intakes. Thus, typical daily intakes of copper would appear to meet nutritional requirements, and would not be expected to result in adverse effects.

However, as with any substance, even essential trace elements, excessive exposures may result in toxicity. There are also certain sensitive sub-populations with genetic defects or other abnormalities in the metabolism of copper that may experience toxicity at levels of exposure that are non-toxic to individuals without these defects. In addition, highly excessive amounts of copper can overwhelm the body's homeostatic regulation of copper intake. Toxicity is likely to occur only when such homoeostatic controls

are overwhelmed and/or basic cellular defense or repair mechanisms are impaired. However, this has only been documented to occur in individuals with genetic copper metabolism impairment (*e.g.,* Wilson's disease, Indian childhood cirrhosis, idiopathic copper toxicosis) or cases of intentional or accidental poisoning, where very large amounts of copper were ingested (ATSDR, 2002; WHO, 1998).

Threshold levels for copper toxicity in humans have not been firmly established. However, it appears that the main intracellular binding site for copper, metallothionein, becomes saturated with copper before toxicity occurs. As metallothionein is believed to act as an intracellular antioxidant which protects cells from free radicals and reactive oxygen species, the saturation of this protein with copper may result in oxidative stress (WHO, 1998). Copper is able to potentially cause oxidative stress through its ability to cycle between an oxidized state, $Cu^{2+}$, and reduced state, $Cu^{+}$. While this ability is key to copper's role in various metalloenzymes, this same property of copper may result in oxidative stress through the generation of superoxide radicals and hydroxyl radicals when converting between the oxidized and reduced states (Camakaris *et al.,* 1999; ATSDR, 2002).

Copper deficiency rarely occurs in humans since most diets have copper in excess of what is required by the body (WHO, 1998). Symptoms of human copper deficiency include anaemia, leucopenia and osteoporosis (ATSDR, 2002). Copper-deficiency is more common in animals, particularly livestock species, and may lead to several different disorders such as anaemia, bone, nerve and cardiovascular disorders, failure of keratinization and reproductive failure (Davis and Mertz, 1987).

Exposure Limits

The following organizations were consulted to select exposure limits for copper: Health Canada; the U.S. EPA; ATSDR; WHO; MOE; JECFA; Health and Welfare Canada; RIVM; and, the National Academy of Science.

***Oral Exposure Limits***

Non-Carcinogenic (Threshold) Effects

The National Academy of Science (IOM, 2001) has derived an acceptable Upper Limit (UL) based on the NOAEL of 10 mg/day based on Pratt *et al*. (1985). IOM (2001) considered this NOAEL to be protective of the general population and felt that no further uncertainty factor was warranted. This decision is supported by the large database of human information indicating no adverse effects in the 10 to 12 mg/day and a paucity of observed liver effects from copper exposure in humans with normal copper homeostasis. This NOAEL results in an acceptable upper limit of approximately 140 µg/kg/day for adults (10 mg/day ÷ 70 kg). There was insufficient data to establish unique ULs for any other age group (similar sensitivity for all ages) (IOM, 2001). Health Canada has indicated that in 2005/2006, the agency will officially adopt ULs as toxicity reference values for all essential elements (Health Canada, 2005 pers. comm. Roest, and Petrovic,) for contaminated sites human health risk assessments.

It is important to recognize that all available regulatory oral exposure limit values for copper are similar in magnitude, and are based on either typical daily intakes, or intakes associated with gastrointestinal distress. Copper doses at, or below any of these values would not be expected to result in adverse health effects under conditions of continuous lifetime daily exposure. It is also important to recognize that all oral exposure limits, regardless of their basis, lie within the range of typical estimated daily dietary intakes, and/or recommended nutritional requirements when the body weight of various human age classes is taken into account (*e.g.*, if a 70 kg adult is assumed, the Health Canada TDI of 0.03 mg/kg body weight/day equates to a daily intake of 2.1 mg Cu/day).

The following is summary of toxicity criteria for copper developed by other agencies.

The Joint Expert Committee on Food Additives (JECFA, 1982) derived a provisional maximum tolerable daily intake (PMTDI) for copper of 0.05 to 0.5 mg/kg body weight/day, which was set equivalent to the provisional daily dietary requirement, rather than an intake associated with an adverse health effect. This value remains as the current PMTDI recommended by JECFA. Health Canada (1996) reported a PTDI of 0.05 to 0.5 mg/kg body weight/day for copper that is based on this PMTDI value.

Health and Welfare Canada (HWC, 1990) estimated safe and adequate dietary copper requirements for a few age classes based on mass balance studies. These safe and adequate requirements were used as the basis for deriving conservative TDIs by Health Canada. For children aged three to 10 years, it was

determined that 0.05 to 0.1 mg/kg body weight/day was the safe and adequate range for daily copper intake. For adults, 0.03 mg/kg body weight/day was estimated as the safe and adequate copper intake rate. These values were used by Health Canada in the derivation of human health soil quality guidelines for copper. Health Canada (2003a) reports an oral TDI of 0.03 mg/kg body weight/day that is based on the adult TDI originally recommended by HWC (1990). In the derivation of the *Guideline for Use at Contaminated Sites in Ontario* (MOE, 1996b), MOE utilized an oral RfD of 30 µg/kg/day for copper.

The U.S. EPA IRIS database contains no oral exposure limits for copper compounds (U.S. EPA, 1988). U.S. EPA Region III (2004), Region VI (2004) and Region IX (2003) all report an oral RfD of 0.04 mg/kg/d, which was originally derived by the U.S. EPA for preparation of the Health Effects Assessment Summary Tables (HEAST). The source of this oral RfD of 0.04 mg/kg body weight/day is as follows. The U.S. EPA (1987) developed a drinking water criterion of 1.3 mg copper/L, based on adverse effects such as vomiting, nausea, and diarrhea in humans following acute consumption of copper in drinking water (as reported in studies by Wyllie, 1957; Semple *et al*., 1960; Chuttani *et al*., 1965). From this drinking water value, U.S. EPA (HEAST) estimated an oral RfD of 0.04 mg/kg/day [(1.3 mg/L x 2 L/d)/70 kg] (ORNL, 2004).

ATSDR (2004) has developed an acute duration oral Minimal Risk Level (MRL) for copper of 0.02 mg/kg/day. This MRL has also been adopted by ATSDR as the intermediate duration MRL. ATSDR (2004) considers that available data are inadequate to derive a chronic duration oral MRL. The acute-duration oral MRL is based on gastrointestinal effects reported in the Pizarro *et al*. (1999) study. To estimate total copper exposure, the dose of copper from drinking water (0.0272 mg Cu/kg/day) in this study was added to the reported average dietary copper intake of copper (0.0266 mg Cu/kg/day). This yielded a total copper exposure level of 0.0538 mg Cu/kg/day, which was considered a NOAEL for gastrointestinal effects. The NOAEL was then divided by an uncertainty factor of three (to account for inter-human variability) to yield the acute oral MRL. ATSDR (2004) notes that this MRL accounts for dietary exposure as well as environmental contamination.

RIVM (Baars *et al*., 2001) noted that copper is an essential nutrient, with a minimum daily requirement of 0.02 to 0.08 mg/kg-day (as reported by WHO, 1996). It was determined that a TDI for copper cannot be lower than the levels required for nutrition essentiality. Thus, RIVM based a TDI on the typical daily intake of the population which was shown to be 0.02 to 0.03 mg/kg/day on average, with a range of 0.003 to 0.1 mg/kg/day and an upper limit of 0.14 mg/kg/day (Slooff *et al*., 1989). This latter upper limit daily intake value (0.14 mg/kg-day) was selected as the TDI by RIVM.

Health Canada (2003b) and the U.S. EPA (2003) have set aesthetic objectives for copper in drinking water of 1.0 mg/L. Aesthetic objectives do not have a toxicological basis, but are established based on objectionable taste, colour and/or staining characteristics. The MOE (2003) has adopted the Health Canada aesthetic objective for copper in drinking water.

For the purposes of this risk assessment an oral RfD of 140 µg/kg/day was selected (IOM, 2001, Health Canada, 2005).

Carcinogenic (Non-threshold) Effects

There is presently no evidence to suggest that copper compounds are carcinogenic in humans or animals (WHO, 1998; ATSDR, 2004; U.S. EPA IRIS, 2004e; TERA, 2004). There are no data available on the genotoxicity of copper in humans exposed *via* oral, inhalation or dermal routes. The existing genotoxicity database suggests that copper is a clastogenic agent, and some studies have shown that exposure to copper can result in DNA damage; however, point mutation assay results are mixed and inconclusive (ATSDR, 2004). Overall, the database on mutagenicity and genotoxicity of copper compounds is limited and equivocal, and considerably more research is required to determine whether or not copper is mutagenic and/or genotoxic to mammals (including humans) *in vivo*.

***Inhalation Exposure Limits***

Non-cancer (Threshold) Effects

RIVM derived a tolerable concentration in air (TCA) of 0.001 mg/m$^3$ based on a NOAEC of 0.6 mg/m$^3$ for lung and immune system effects in rabbits from a short-term toxicity study by Johansson *et al*., (1984). RIVM used an uncertainty factor of 100 (10 each for intra- and interspecies variability), and adjusted for continuous exposure (5/7 x 6/24) to yield the TCA.

OEHHA (1999) derived an acute reference exposure level (REL) for a one hour exposure of 0.1 mg/m$^3$. This acute REL is considered protective against mild adverse effects. The REL was derived based on studies by Gleason (1968), and Whitman (1957; 1962) which investigated metal fume fever in workers. A NOAEL of 1 mg/m$^3$ was identified from these studies. The NOAEL was mainly based on the report of Whitman (1957) indicating that exposure to copper dust was detectable by taste, but that no other symptoms occurred following exposure to 1 to 3 mg/m$^3$ for an unspecified short duration. Given that the exposure duration was not clearly stated in these studies, no extrapolation to a one hour concentration could be conducted. Rather, the NOAEL was assumed to be applicable to a one hour exposure. A

cumulative uncertainty factor of 10 was applied to the NOAEL (for intraspecies uncertainty) to yield the acute REL. Given the limitations of the existing data, OEHHA suggests that re-evaluation of the acute REL for copper be conducted when better methods or data are available. OEHHA did not derive a chronic REL for inhalation exposure to copper.

The MOE (2005b) reports a 24-hr Ambient Air Quality Criterion (AAQC) of 50 µg/m$^3$ for copper, based on health concerns. No supporting rationale for this AAQC was identified in available MOE publications.

ATSDR (2004) considers available data on the toxicity of inhaled copper inadequate for derivation of acute, intermediate, or chronic duration inhalation MRLs.

The U.S. EPA has not derived inhalation exposure limits for any copper compound (U.S. EPA, 1988).

For the purposes of this risk assessment an inhalation TCA of 1 µg/m$^3$ derived by RIVM (Baars *et al*., 2001) was selected.

Cancer (Non-threshold) Effects

There is presently no evidence to suggest that copper compounds are carcinogenic in humans or animals (WHO, 1998; ATSDR, 2004; U.S. EPA IRIS, 2004e; TERA, 2004). There are no data available on the genotoxicity of copper in humans exposed via oral, inhalation or dermal routes.

***Dermal Exposure Limits***

No regulatory dermal exposure limits for copper compounds were identified in the literature reviewed for the current assessment. Route-to-route extrapolation was used to derive an appropriate limit for the current assessment.

## Lead

Essentiality

Lead is not known to be an essential micronutrient in humans or other mammals.

Exposure Limits

As described below WHO, Health Canada, RIVM, MOE, ATSDR, U.S. EPA and OEHHA were the organizations consulted to select exposure limits for lead.

Although the toxicological database for lead is large, the majority of human effects data are expressed as a blood lead (PbB) concentration, rather than a dose or concentration in an environmental medium. In addition, there are inadequate empirical data for demonstrating a threshold for the health effects of lead. . In fact, many consider lead a non-threshold toxicant, indicating that any expsoure to lead leads to possible efects. Given these limitations, many regulatory agencies have not derived conventional exposure limits such as RfDs, TDI's or MRLs, and advocate that exposure to lead should be minimized. In order to utilize the wealth of literature relating human PbB concentrations to health effects, such agencies (*e.g.*, ATSDR, U.S. EPA) have developed models or other approaches to relate environmental lead exposure to PbB levels. This is described further in Section 5.0. In addition, environmental quality guidelines for lead have also been developed with a different approach than is used for most other chemicals. Instead of developing exposure limits based on no- or low-effects-levels observed in test organisms following controlled exposures, lead guidelines are typically back calculated from a critical PbB concentration (usually 10 µg/dL, as recommended by CDC, 2004, 2005; U.S. EPA, 2004d CEOH, 1994). A blood lead level of ≥10 µg/dL is a level of concern in an individual and is indicative of elevated exposure and possible harm to health.

Although recent scientific data indicate an association between intellectual performance in children and PbB levels < 10 µg/dL, it appears that major agencies (MOE, 2006; U.S. EPA, 2002, 2006, 2007; CDC, 2004, 2005) acknowledge that a clear threshold for protection of neurological impacts in children has not yet been identified. In addition, derivation of acceptable exposure levels is complicated by numerous confounding factors that influence lead toxicity, including socioeconomic status, pre-existing lead body burdens, age, health status, nutritional status and lifestyle factors such as alcohol consumption and tobacco smoke (environmental tobacco smoke has been associated with elevated PbB). As a result, CDC (2004; 2005) has recommended that the PbB level of concern should remain at their 1991 (CDC, 1991)

recommended level of 10 µg/dL. The decision for not lowering the PbB level of concern below10 µg/dL is based on the following (CDC, 2004; 2005):

- Lack of effective clinical interventions to lower PbB levels for children with levels less than 10 µg/dL or to reduce the risks for adverse developmental impacts;
- Inaccuracy inherent in laboratory analytical testing of PbB levels in children; and,
- No evidence of clear threshold for neurological impacts in children and as such, a decision to lower the PbB level of concern would be "arbitrary" and "provide uncertain benefits".

Health Canada is currently undergoing a review of their PbB population intervention level established by CEOH in 1994.

***Oral Exposure Limits***

Non-Carcinogenic (Threshold) Effects

While the issue of whether or not a threshold exists for the cognitive effects of lead in children continues to be debated, there is consistent information from the available lead health effects literature indicating that PbB levels > 10 µg/dL are linked to decreased intelligence and impaired neurobehavioral development (Lanphear *et al*., 2005; CDC, 2004; CDC, 2005; U.S. EPA, 2004d; ATSDR, 2005; WHO, 1995) in children.

The MOE (1994) recommended an intake of concern for populations ($IOC_{pop}$) of 1.85 µg/kg/day in order to minimize the predicted number of children with individual blood lead levels of concern. Subclinical neurobehavioural and developmental effects were the critical effects appearing at the lowest levels of exposure (MOE, 1994). The intake of concern for individuals ($IOC_{ind}$) was based on a Lowest Observed Adverse Effect Level (LOAEL) in infants and young children of 10 µg/dL PbB divided by an intake/PbB slope factor of 0.21 µg Pb per DL PbB per µg/day. This resulted in an $IOC_{ind}$ of 3.7 µg/kg/day for a 13 kg child (0.5-4 yrs). To derive the $IOC_{pop}$ an uncertainty factor of 2 was applied to the $IOC_{ind}$, which resulted in a daily intake of 1.85 µg/kg/day (MOE, 1994). This value is based on the same research as the other agencies limits, as described below. In the *Guideline for Use at Contaminated Sites in Ontario* (MOE, 1996a), MOE adopted the oral RfD of 1.85 µg/kg/day for lead. As it is based on an internal PbB concentration, this $IOC_{pop}$ applies to lead exposure received from all sources, *via* all routes.

A value that is the basis for many jurisdiction's exposure limits is the TDI of 3.57 µg/kg/day derived by the World Health Organization. The TDI was derived based on a PTWI of 25 µg of lead per kg of body weight recommended by FAO/WHO (1993), and reaffirmed by WHO (1999), for all age groups. This PTWI value was in turn based upon technical reports presented at annual meetings of the Joint FAO/WHO Expert Committee on Food Additives (JECFA), as well as upon epidemiological studies associating lead exposure with neurological effects in infants and children. The PTWI represents the permissible or tolerable human weekly intake that is unavoidable as a result of consuming typical foods. The PTWI was based on metabolic studies of infants which indicated that if daily lead intakes equal approximately 5 µg/kg/day then PbB levels remained at a fairly constant low level within the body. Daily intakes of lead in the range of 3-4 µg/kg body weight/day by infants and children were not associated with an increase in blood lead concentrations (WHO, 1995; Ziegler *et al.*, 1978; WHO, 1987). The PTWI of 25 µg/kg/week was converted to a PTDI of 3.57 µg/kg/day by dividing the PTWI by seven (for seven days in a week). The TDI for lead accounts for exposure from all sources and is considered protective of all humans, including infants and children.

Health Canada (2003a) has adopted 3.6 µg/kg body weight/day as the provisional TDI for lead, and the CCME and Health Canada use this value as the basis for derivation of soil and drinking water guidelines that are protective of human health. In the Netherlands, RIVM (Baars *et al.*, 2001) has also derived a TDI of 3.57 µg/kg body weight/day, based on the PTWI of 25 µg/kg/week derived by the FAO/WHO (1993).

Two regulatory agencies that are typically leaders in the development of chemical exposure limits (ATSDR and U.S. EPA) have not derived any exposure limits for lead compounds; rather, they have developed alternate approaches that relate environmental lead exposure to PbB levels.

ATSDR (2005) did not derive any minimal risk levels (MRLs) for lead due to the lack of a clear threshold for health effects and the need to consider multi-media routes of exposure. However, ATSDR has developed guidance for employing media-specific slope factors to integrate exposures from various pathways for site-specific risk assessments.

The U.S. EPA IRIS database does not recommend oral or inhalation reference doses (or concentrations) for lead due to high levels of uncertainty, and because lead is considered a non-threshold toxicant (U.S. EPA, 2004d). The U.S. EPA believes that the effects of lead exposure, particularly changes in blood enzyme levels, and children's neurodevelopment, may occur at blood levels so low as to be essentially without a threshold. As further support for not deriving an RfD or RfC, the U.S. EPA (2004d) states that current knowledge of lead toxicokinetics suggests that risk values derived by standard procedures (such as

an oral RfD) would not be representative of the potential risk, due to difficulties in attempting to account for pre-existing body burdens of lead, and certain life-stages when stored lead may be mobilized within the body. For example, lead is well known to be stored in bone tissue, and its mobilization from bone varies greatly with age, health status, nutritional state, physiological state (pregnant, lactation, menopause *etc*.). Alternatively, the U.S. EPA has developed the Integrated Exposure Uptake Biokinetic Model (IEUBK) as a means of predicting the occurrence of blood lead concentrations above 10 µg/dL in children. This model is used to determine the contribution of lead from all media to PbB (U.S. EPA, 2004d). The IEUBK model predicts the geometric mean PbB concentration for a child exposed to lead in various media (or a group of similarly exposed children). The model can also calculate the probability that the child's PbB exceeds 10 µg Pb/dL (P10). Preliminary remediation goals (SRMLs) for lead are generally determined with the model by adjusting the soil concentration term until the P10 is below a 5% probability (U.S. EPA, 2003). In addition, an Adult Lead Model was developed by the U.S. EPA Superfund Program for when adult exposures to lead are of concern, especially in the case of pregnant women (www.epa.gov/superfund/programs/lead/adult.htm). The model equations were developed to calculate cleanup goals such that there would be no more than a 5% probability that fetuses exposed to lead would exceed a blood lead (PbB) of 10 µg/dL.

For the purposes of this risk assessment an oral, inhalation and dermal exposure limit of 1.85 µg/kg/day was selected for lead (MOE, 1996a, MOE, 1994).

Carcinogenic (Non-threshold) Effects

The U.S. EPA (2004d) has classified lead compounds as B2 - probable human carcinogen, based on sufficient animal evidence of kidney tumours, but inadequate human evidence. The U.S. EPA has determined that an estimate of carcinogenic risk from oral exposure (such as a slope factor) using standard methods would not adequately describe the potential risk for lead compounds. The U.S. EPA's Carcinogen Assessment Group made this determination given the current lack of understanding on various toxicological and toxicokinetic characteristics of lead.

IARC (2004) classified inorganic lead compounds as probably carcinogenic to humans (Group 2A), based on limited evidence for carcinogenicity in humans and sufficient evidence for carcinogenicity in experimental animals. The IARC evaluation considers the evidence of carcinogenicity in humans and experimental animals, as well as other data relevant to the evaluation of carcinogenicity and its mechanisms. For example, IARC (2004) noted that while there appears to be little evidence that lead is directly genotoxic, it may be indirectly genotoxic as a result of oxidative stress effects caused by the

formation of reactive oxygen species. The IARC Working Group does not typically provide quantitative estimates of any chemical's carcinogenic risk.

Health Canada has not formally classified lead compounds with respect to their carcinogenic potential.

The OEHHA (California Environmental Protection Agency Office of Environmental Health Hazard Assessment) considers lead compounds human carcinogens as they have derived both oral and inhalation slope factors and unit risks for lead (see Appendix A4 for details). However, at this time, no other regulatory agencies, other than OEHHA, are known to have derived regulatory exposure limits for lead that are based on carcinogenic effects.

***Inhalation Exposure Limits***

Non-cancer (Threshold) Effects

There are no non-cancer inhalation exposure limits for lead. However, the FAO/WHO TDI of 3.57 µg/kg/d accounts for lead exposure from all sources and is considered protective of all humans, including infants and children. The MOE $IOC_{pop}$ of 1.85 µg/kg/day is also considered protective of multimedia lead exposure (MOE, 1994). Because the $IOC_{pop}$ proposed by the MOE (1994) was intended for all routes of exposure, 1.85 µg/kg/day was selected as the inhalation RfD and the oral RfD for the current assessment.

The Ontario Ministry of the Environment (MOE, 2007) derived a 24-hour Ambient Air Quality Criterion (AAQC) of 0.5 µg/m$^3$ and a half-hour Point of Impingement (POI) Limit of 6.0 µg/m$^3$ for lead based on health effects. The MOE has summarized the scientific basis for air quality guidelines and standards developed by the U.S. Environmental Protection Agency (US EPA), the California Environmental Protection Agency (Cal/EPA), New Zealand, Australia, United Kingdom and the World Health Organization. Following this review, MOE selected the Cal/EPA's (2001) derivation of their lead air guideline as the most appropriate approach on which to base an updated air standard for lead in Ontario (24-hour AAQC of 0.5 µg/m$^3$). Cal/EPA's guideline is based on an air concentration associated with a 5% probability of exceeding the BLL of concern. The MOE (2005b) also reported AAQCs for lead in dustfall of 0.1 g/m$^2$ over 30 days.

For the purposes of this risk assessment an oral, inhalation and dermal exposure limit of 1.85 µg/kg/day was selected for lead (MOE, 1996a, MOE, 1994).

Cancer (Non-threshold) Effects

Only one agency was identified as having developed quantitative toxicity estimates based on the carcinogenicity of lead (*i.e.*, OEHHA, 2002). The U.S. EPA did not derive any exposure limits based on carcinogenic endpoints as its Carcinogen Assessment Group concluded that the uncertainties associated with lead pharmacokinetics and factors affecting the absorption, release, and excretion of lead (*i.e.*, age, health, nutritional status, body burden, and exposure duration) preclude the development of a numerical estimate to predict carcinogenic risk (U.S. EPA, 2004d). Thus, the U.S. EPA believes that the current limited knowledge of lead toxicokinetics suggests that a carcinogenic risk estimate derived by standard procedures would not adequately represent the true potential risk.

The OEHHA (2002) reports an inhalation unit risk factor of 1.2 E-5 $(\mu g/m^3)$-1, an inhalation slope factor of 0.042 $(mg/kg/day)^{-1}$, and oral slope factor of 0.0085 $(mg/kg/day)^{-1}$. All values were originally calculated by OEHHA (1997) from rat kidney tumor incidence data (the Azar *et al*., 1973, study) using a linearized multistage procedure. The studies by Azar *et al*, (1973) and Koller *et al*. (1985) were considered to represent the best available tumour dose-response data for use in quantitative cancer risk assessment. The derivation of the unit risk and slope factors is described in detail within OEHHA (2002). In summary, rat kidney tumour data were extrapolated to humans by means of the best fitting linearized multistage model (*i.e.*, GLOBAL86), conversion of rat doses to human equivalent doses (HED), the use of standard human receptor parameters, and assumptions related to the inhalation and oral bioavailability of lead.

For the purpose of the current assessment, lead was not evaluated as a carcinogen for oral or inhalation exposures.

***Dermal Exposure Limits***

No regulatory dermal exposure limits for lead compounds were identified in the literature reviewed for the current assessment.

For the purposes of this risk assessment an oral, inhalation and dermal exposure limit of 1.85 µg/kg/day was selected for lead (MOE, 1996a, MOE, 1994).

## Nickel

Essentiality

Nickel is an essential trace element in animals, based on reports of nickel deficiency in several animal species (*e.g.*, rats, chickens, cows, and goats) (ATSDR, 2003). Effects of nickel deficiency were observed in the liver and included abnormal cellular morphology, oxidative metabolism, and increases and decreases in lipid levels. In addition, decreases in growth and haemoglobin concentrations and impaired glucose metabolism have also been observed. However, nickel deficiency has never been reported in humans as nickel intake generally exceeds dietary requirements (Anke *et al.*, 1995; Denkhaus and Salnikow, 2002). Nickel is widely considered to be a normal constituent of the diet, with daily intakes ranging from 100 to 300 µg/day (U.S. EPA, 1991a). The functional importance of nickel has not been clearly demonstrated as no enzymes of cofactors that include nickel are known in humans (Denkhaus and Salnikow, 2002). Therefore, the essentiality of nickel in humans has not been confirmed and nickel dietary recommendations have not been established for humans (ATSDR, 2003; Denkhaus and Salnikow, 2002).

Exposure Limits

TERA, ATSDR, OEHHA, Health Canada, the U.S. EPA were consulted to select exposure limits for nickel. In addition, the inhalation exposure limits developed by Seilkop (2004) were also reviewed as they were considered this study given the current state of knowledge related to nickel compounds and their toxicological behaviour.

### *Oral Exposure Limits*

Non-Carcinogenic (Threshold) Effects

Health Canada (1996; 2003a) reported a TDI of 50 µg Ni/kg/day for nickel sulfate. This value was derived from a NOAEL of 5 mg/kg/day reported by Ambrose *et al.*, (1976) for a two year dietary study in which rats were administered nickel sulphate hexahydrate. The critical effects observed in the chronic oral study included decreased body and organ weights. An overall uncertainty factor of 100 (10-fold for interspecies extrapolation and 10-fold for interspecies variation) was applied to the study NOAEL (considered the human-equivalent NOAEL) to yield the TDI. OEHHA (2003) used the same principal study (*i.e.*, Ambrose *et al.*, 1976) to derive a chronic oral reference exposure level (REL) of 50 µg/kg/day.

The U.S EPA (1991b) reported an oral RfD of 20 µg/kg/day for nickel which was also based on the chronic oral rat study by Ambrose *et al*. (1976). The NOAEL was then adjusted by an uncertainty factor of 300; 10 for interspecies extrapolation, 10 to protect sensitive populations, and an additional factor of three to account for inadequacies in the reproductive studies which was not included in the derivation of the Health Canada TDI. The two-year feeding study in rats was supported by a subchronic gavage study in water (ABC, 1986), which indicated the same NOAEL of 5 mg/kg/day. The oral RfD of 20 µg/kg/day was adopted for this risk assessment.

Recently, in their review of the toxicology of soluble nickel compounds, TERA (2004) calculated an oral reference dose of 8 µg/kg/day nickel for ingested nickel-soluble salts. The most sensitive endpoint was determined to be increased albuminuria (indicating renal glomerular dysfunction) in male and female rats exposed to nickel in drinking water for six months (Vyskocil *et al*., 1994a;b). An overall uncertainty factor of 1,000 was applied (*i.e*., 10 for intrahuman variability, 10 for interspecies extrapolation, and a 10 for subchronic-to-chronic extrapolation, an insufficient toxicological database, and use of a minimal LOAEL) to a LOAEL of 7.6 mg/kg/day to yield the oral RfD of 8 µg/kg body weight/day.

TERA (2004) notes that the nickel doses used in the principal study did not include the nickel present in the diet. Therefore, the RfD represents the dose of nickel in addition to the amount received in food. TERA (2004) considers this oral RfD to agree well with the U.S. EPA oral RfD of 20 µg/kg body weight/day for total nickel exposure, and is within the expected inherent uncertainty surrounding an RfD. In addition, an independent peer review panel, through TERA's ITER Peer Review program has recently approved the oral RfD value. The TERA RfD was not used in this study as reference values derived for all other COC were expressed on a total exposure basis, whereas this value is considered an incremental value.

For consistency purposes, the U.S. EPA value of 20 µg/kg/day was selected for the purposes of this risk assessment.

Carcinogenic (Non-threshold) Effects

There are no data available on the carcinogenicity of nickel in humans exposed *via* oral or dermal routes.

***Inhalation Exposure Limits***

Non-cancer (Threshold) Effects

Health Canada (1996) recommended various guidance values for inhalation exposure to different forms of nickel. A tolerable inhalation concentration (non-cancer effects) of 0.0035 µg/m$^3$ was recommended for nickel sulfate based on a study of lung and nasal lesions in rats and mice observed by Dunnick *et al.* (1989). Tolerable inhalation concentrations of 0.018 µg/m$^3$ and 0.02 µg Ni/m$^3$ were recommended for metallic nickel, and nickel oxide, respectively.

OEHHA (2003) derived a chronic reference exposure level (REL) of 0.00005 mg/m$^3$ for nickel compounds (except nickel oxide). The principal study was NTP (1994b) and the critical effects were pathological changes in lung, lymph nodes, and nasal epithelium, which included active pulmonary inflammation, macrophage hyperplasia, alveolar proteinosis, fibrosis, lymph node hyperplasia, and olfactory epithelial atrophy. The study NOAEL was 0.03 mg/m$^3$. The nickel species tested was nickel sulfate hexahydrate. The study NOAEL was adjusted for continuous exposure (multiplied by 6/24 x 5/7) and then converted to a NOAEL HEC (human equivalent concentration) by multiplying against a regional deposited dose ratio (RDDR) of 0.29. Following this, the $NOAEL_{HEC}$ was divided by a cumulative uncertainty factor of 30 (three for interspecies uncertainty; 10 for intraspecies uncertainty) to yield the chronic REL.

OEHHA (2003) derived a chronic REL specifically for nickel oxide of 0.0001 mg/m$^3$. The principal study was NTP (1994c), and the critical effects considered were pathological changes in lung and lymph nodes, including active pulmonary inflammation, lymph node hyperplasia, and adrenal medullary hyperplasia (females only). This study identified a LOAEL of 0.5 mg/m$^3$. The study NOAEL was adjusted for continuous exposure (multiplied by 6/24 x 5/7) and then converted to a $NOAEL_{HEC}$ by multiplying against an RDDR of 0.29. Following this, the NOAELHEC was divided by a cumulative uncertainty factor of 300 (*i.e.,* 10 for use of a LOAEL, three for interspecies uncertainty, and 10 for intraspecies uncertainty) to yield the chronic REL.

The exposure limits derived by OEHHA (2003) were selected for use in this study.

Cancer (Non-threshold) Effects

Oller (2002) suggested that in isolation, water soluble nickel compounds are not complete carcinogens. However, they may enhance the carcinogenic risks associated with other compounds when inhaled, if concentrations are large enough to induce chronic lung inflammation (Oller, 2002). By keeping exposure below levels resulting in chronic respiratory toxicity, Oller (2002) suggested that possible tumour-enhancing effects would be avoided. Seilkop (2004) has derived an inhalation unit risk of $1.9x10^{-4}$ $(\mu g/m^3)^{-1}$ for nickel sulphate when exposure is in the presence of a carcinogen. The basis of this unit risk is the incidence of an inflammatory response in the exposed animals. While there is unquestionably a link between inflammation and cancer promotion, the use of this endpoint in the derivation of a unit risk is uncertain. As such, this unit risk has not been utilized in this assessment, as clear evidence exists to indicate that nickel sulphate act *via* a non-mutagenic mechanism.

Seilkop and Oller (2003) have estimated safety limits for workers from fitted animal dose-response curves after accounting for interspecies differences in deposition and clearance, differences in particle size distributions, and human work activity patterns. Using a $10^{-4}$ risk level (which they deemed an acceptable occupational lifetime cancer risk level), they derived an occupational exposure limit concentration of 0.002 to 0.01 mg inhalable nickel subulphide/$m^3$. Subsequently, Seilkop (2004) has derived an inhalation unit risk of $6.3x10^{-4}$ $(\mu g/m^3)^{-1}$ for nickel subsulphide. This IUR is independent of any specific risk level and can be used to estalish $10^{-5}$ or $10^{-6}$ acceptable risk levels as appropriate.

Seilkop and Oller (2003) also estimated safety limits for workers from fitted animal dose-response curves. Using a $10^{-4}$ risk level (which they deemed an acceptable occupational lifetime cancer risk level), they derived an occupational exposure limit concentration of 0.5 to 1.1 mg inhalable nickel oxide/$m^3$. The authors report that although the animal data for nickel oxide suggest a threshold response for lung cancer, this cannot be concluded with certainty as sampling uncertainty in data make the non-threshold response equally as plausible. They report that the non-linearity of the observed dose-response of nickel oxide is well represented by benchmark dose models (excluding the high dose response). Subsequently Seilkop (2004) has derived an inhalation unit risk of $2.3 x10^{-5}$ $(\mu g/m^3)^{-1}$ for nickel oxide.

Interestingly, when the IURs for nickel oxide and nickel sulphate (Seilkop, 2004) are converted to a risk-specific concentration (RsC) assuming a target risk level of one-in-one hundred thousand, the RsCs are very similar to the OEHHA chronic RELs for both 'nickel compounds except nickel oxide' and nickel oxide. The RsC that corresponds to the nickel sulphate IUR is 0.05 $\mu g/m^3$ (the OEHHA REL is also 0.05 $\mu g/m^3$), while the RsC that corresponds to the nickel oxide IUR is 0.4 $\mu g/m^3$ (the OEHHA REL is

0.1 µg/m$^3$). Thus, it can be extrapolated from this comparison that although the OEHHA chronic RELs are not derived from a cancer endpoint, they would appear to be protective of both non-cancer and potential cancer effects of inhaled soluble nickel and nickel oxide. However, the OEHHA chronic RELs are not protective of the potential carcinogenic effects of nickel subsulfide, based on converting the Seilkop (2004) IUR for this substance to an RsC at a one-in-one hundred thousand target risk level.

Following an detailed evaluation of three different mechanistic approaches, an EU working group proposed a limit value range of 0.01 to 0.05 µg Ni/m$^3$ (as an annual mean), based upon non-cancer effects. This working group also believed that a limit value in this range can be judged compatible with the objective of limiting excess lifetime cancer risks to not more than one-in-a-million. The majority of the working group proposed a limit value at the lower end of this range, to represent an annual mean of total airborne nickel (European Commission DG Environment, 2001).

Based upon this work, in 2004 the European Parliament adopted a target value for airborne nickel of 20 ng Ni/m$^3$, or 0.02 µg Ni/m$^3$, considered protective of both cancer and non-cancer health endpoints (OJEU, 2005). This value was selected as the primary exposure limit for nickel inhalation used in this risk assessment.

Health Canada (1996; 2003a) considers oxidic, sulphidic and soluble nickel to be carcinogenic to humans. A Tumorigenic Concentration 05 ($TC_{05}$) of 70 µg/m$^3$ has been developed by Health Canada (1996) for soluble nickel (primarily nickel sulphate and nickel chloride) based on lung cancer mortality observed in a cohort in Norway (Doll *et al.*, 1990). A $TC_{05}$ of 40 µg/m$^3$ was recommended for combined oxidic, sulphidic and soluble nickel. Assuming that a 70 kg person breathes at a rate of 20 m$^3$/day, the inhalation slope factors for soluble nickel and combined oxidic, sulphidic and soluble nickel were estimated to be 0.0025 (µg/kg/day)$^{-1}$ and 0.0044 (µg/kg/day)$^{-1}$, respectively.

The World Health Organization (WHO, 2000) developed an incremental unit risk of 0.0004 (µg/m$^3$)$^{-1}$ for nickel subsulphide based on epidemiological lung cancer data for nickel refinery workers (Chovil *et al.*, 1981; Magnus *et al.*, 1982; Doll, 1977). WHO (2000) reassessed this unit risk based on updated epidemiology data for lung cancer in refinery workers, including follow-up studies of a cohort examined in Kristiansand, Norway used in the 1987 assessment (Andersen, 1992; Andersen *et al.*, 1996). Based on the estimated risk for this cohort of 1.9 and a lifetime exposure estimate of 155 µg/m$^3$, the WHO (2000) calculated an incremental life-time unit risk of 0.00038 (µg/m$^3$)$^{-1}$ for inhalation of nickel. This unit risk of 0.00038 (µg/m$^3$)$^{-1}$ was then converted to an inhalation cancer slope value of 0.00133 (µg/kg/day)$^{-1}$ based on an adult body weight of 70 kg and a breathing rate of 20 m$^3$/day.

A cohort of employees of a nickel refinery in West Virginia who experienced a minimum one year exposure to nickel refinery dusts (containing nickel subsulphide, sulphate and oxide or only nickel oxide) did not show an increased incidence of lung cancer above expected rates (Enterline and Marsh, 1982). Chovil *et al.* (1981) studied a cohort of nickel refinery workers in Ontario, and observed a dose-related trend for the relationship between weighted exposure in years to the incidence of lung cancer. Similarly, a cohort of Welsh nickel refinery workers had elevated risks of cancer compared to the national average; increased rates of nasal cancer were observed in men employed prior to 1920, while this rate was less than the national average for those starting work between 1920 and 1925, and equalled the expected value for those employed after 1925 (Doll *et al.*, 1977). A significantly increased lung cancer-related mortality was observed in employees starting prior to 1925 but not in those starting between the years 1930 to 1944. Magnus *et al.* (1982) conducted a study of men employed at a nickel refinery in Norway, and reported an elevated occurrence of respiratory cancer for nickel- exposed workers compared to expected values, and for workers involved in nickel processing steps compared to non-processing employees.

Each of these epidemiology studies used in the U.S. EPA determination of the unit risk associated with nickel, had factors limiting their usefulness for a unit risk calculation. For example, none were able to account for exposures to other chemicals, metals or nickel species (such as nickel subsulphide), that were present in the occupational environment of a nickel refinery. Only one attempted to account for differences between smokers and non- smokers, an important consideration when examining the incidence of lung cancer. Three of the four studies did not provide measurements of airborne nickel concentrations or estimates of worker exposure. The U.S. EPA estimated exposures based on information provided in other reports in which concentrations of nickel in the work environment were projected on the basis of the operating procedures used. Other problems included poorly or heterogeneously defined cohorts, poor follow-up success, and no consideration of the role of the latency period for lung cancer. Of the four summarized above, Enterline and Marsh (1982) was the most relevant since estimated exposures were provided, the latent period could be examined, and the effects in refinery workers could be compared to non-refinery workers. However, the mixed exposure to other substances and to cigarette smoke were confounding factors that limit the interpretation of that study.

Based on these studies, the U.S. EPA derived slope factors for nickel refinery dust and nickel subsulphide of $2.4 \times 10^{-4}$ $(\mu g/m^3)^{-1}$ and $4.8 \times 10^{-4}$ $(\mu g/m^3)^{-1}$, respectively. These slope factors are not directly attributed to the Sudbury environment given the concomitant presence of multiple nickel species in proportions that will differ from those found within refineries. However, these slope factor were used in a weight of

evidence approach most notably for consideration of the potential presence of nickel sulphide in the ambient environment.

***Dermal Exposure Limits***

No regulatory dermal exposure limits for nickel compounds were identified in the literature reviewed for the current assessment. Route-to-route extrapolation was used to derive an appropriate limit for the current assessment.

## Selenium

Essentiality

There is widespread scientific consensus that selenium is an essential trace element in both animal and human nutrition (NAS, 1976; Bennett, 1982; WHO, 1986; Levander, 1982; Robinson, 1982; Foster and Sumar, 1997). Selenium has even been reported to be an essential trace element in all vertebrates (Bowen, 1979).

Selenium deficiency in isolation seldom causes overt illness; however it leads to biochemical changes that predispose selenium-deficient individuals to illness associated with other stresses. (NAS, 2000). A deficiency of selenium in the human diet is associated with Keshan disease (juvenile cardiomyopathy endemic to certain areas of China) and Kashin-Beck disease (osteoarthropathy endemic to Eastern Siberia) as well as numerous other diseases, conditions and effects (Chen *et al.,* 1980; Sokoloff, 1985; Wu *et al.,* 1989).

Selenium is a rather unique element in that there is a small margin of safety (ranging from a factor of approximately 5 to 18 between levels of selenium compounds that constitute dietary deficiency and those that result in toxicity (Lemly, 1997). The U.S. National Academy of Sciences (NAS, 2000) has recommended a safe and adequate daily intakes ranging from 20 to 70 µg per person per day for adults (Table 4.28). The estimated average requirements for selenium were extrapolated from human balance studies and laboratory animal studies (NAS, 2000). The RDAs for selenium of 70 µg/day for adult men and 70 µg/day for adult women were based on a daily dose 0.87 µg/kg bw/day derived from a series of depletion studies carried out in Chinese males (Yang *et al.,* 1989a,b; Yang *et al.,* 1988; Levander, 1991). RDAs for children and infants were extrapolated from the adult RDAs on the basis of body weight. For children, the RDA is set at 0.87 µg/kg bw/day (NAS, 2000). Selenium dietary requirements for pregnant

or lactating mothers are greater, with RDAs of 60 and 70 µg/day respectively (NAS, 2000). The dietary requirement below which adverse human health effects resulting from deficiency may occur has been tentatively estimated to range from 2 to 120 µg/day (Stewart *et al.,* 1978). Whanger *et al.,* (1996) suggest that an intake of less than 40 µg/day will likely result in deficiency. The minimum dose to cause toxicity in humans is not well defined, but the threshold appears to lie in the range of 400 to 900 µg/day (Allegrini *et al.,* 1985; Yang *et al.,* 1989a; 1989b; Longnecker *et al.,* 1991; Whanger *et al.,* 1996).

**Table 4.28 Recommended Allowable Intakes for Selenium[a]**

| Age | Intake/day AI or EAR[b] | RDA[b] |
|---|---|---|
| Selenium AI: Summary, Ages 0 through 12 months | | |
| 0 to 6 months | 15 µg (0.19 µmol)/day | ≈ 2.1 µg/kg |
| 7 to 12 months | 20 µg (0.25 µmol)/day | ≈ 2.2 µg/kg |
| Selenium EAR/RDA: Children and Adolescents ages 1 through 18 years | | |
| Children 1 to 3 years | 17 µg (0.22 µmol)/day | RDA = 20 µg/day |
| Children 4 to 8 yrs | 23 µg (0.29 µmol)/day | RDA = 30 µg/day |
| Boys and Girls 9 to 13 yrs | 35 µg (0.45 µmol)/day | RDA = 40 µg/day |
| Boys and Girls 14 to 18 yrs | 45 µg (0.57 µmol)/day | RDA = 55 µg/day |
| Selenium EAR/RDA: Adults 19 through 50 yrs | | |
| Men and women 19 to 30 yrs | 45 µg (0.57 µmol)/day | RDA = 55 µg/day |
| Men and Women 31 to 50 yrs | 45 µg (0.57 µmol)/day | RDA = 55 µg/day |
| Selenium EAR/RDA: Adults >50 yrs | | |
| Men and Women 51 to 70 | 45 µg (0.57 µmol)/day | RDA = 55 µg/day |
| Men and women >70 yrs | 45 µg (0.57 µmol)/day | RDA = 55 µg/day |
| Selenium EAR/RDA: Women during and after pregnancy | | |
| Pregnant women 14 to 18 yrs | 49 µg (0.62 µmol)/day | RDA = 60 µg/day |
| Pregnant women 19 to 30 yrs | 49 µg (0.62 µmol)/day | RDA = 60 µg/day |
| Pregnant women 31 to 50 yrs | 49 µg (0.62µmol)/day | RDA = 60 µg/day |
| Lactating women 14 to 18 yrs | 59 µg (0.75µmol)/day | RDA = 70 µg/day |
| Lactating women 19 to 30 yrs | 59 µg (0.75µmol)/day | RDA = 70 µg/day |
| Lactating women 31 to 50 yrs | 59 µg (0.75µmol)/day | RDA = 70 µg/day |

[a] Adapted from NAS, 2000.
[b] EAR = estimated average requirement; RDA = recommended dietary allowance.

Various studies have indicated that supplementary selenium relieves a number of human health problems. These include muscular discomfort, cardiomyopathy, arthritis, cataracts, cystic fibrosis, hemolytic anemia, multiple sclerosis, Kwashiorkor (a protein-calorie malnutrition), night blindness, and immunodeficiencies (Foster and Sumar, 1997; van Rij *et al.,* 1979; Johnson *et al.,* 1981). Furthermore, selenium is an essential component of glutathione peroxidase (GSHPx), an enzyme which protects cell

membranes from oxidative damage, and type 1 iodothyronine 5`-deiodinase, an enzyme which interacts with iodine to prevent abnormal thyroid hormone metabolism (Foster and Sumar, 1997).

Selenium is also believed to have a protective function against certain types of cancers (Foster and Sumar, 1997). Levander (1987) hypothesized that the "anti-cancer" protective effects of selenium are due to its roles in alleviating oxidative damage, altering carcinogen metabolism, and selective toxicity against rapidly dividing tumour cells. It should be noted that there is conflicting evidence with respect to this function of selenium. Nonetheless, relatively high levels of selenium have been used successfully to protect against both chemically-induced and spontaneously occurring tumours in laboratory animals (Combs and Combs, 1986; Ip and Ganther, 1992; Whanger, 1983). Selenium supplementation has also been shown to significantly inhibit tumours induced by viruses, or ultraviolet radiation (ATSDR, 2003). Methylated forms of selenium appear to be the most important with respect to cancer prevention.

Exposure Limits

The following organizations were consulted to select exposure limits for selenium: the U.S. EPA; ATSDR; HC; National Academy of Science; MOE; and, OEHHA.

***Oral Exposure Limits***

Non-Cancer (Threshold) Effects

In determining the oral RfD for selenium compounds, the U.S. EPA selected the epidemiology study by Yang *et al.* (1989) as the principal and supporting study. The study NOAEL of 15 µg/kg bw/day was used to calculate an oral RfD of 5 µg/kg bw/day; a three-fold uncertainty factor was applied to account for sensitive individuals (U.S. EPA, 1991a). A 10-fold uncertainty factor was not deemed necessary because of the high level of confidence in the Yang *et al.* (1989) and additional supporting studies (U.S. EPA, 1991a). The results of Longnecker *et al.* (1991) strongly corroborate the NOAEL identified by Yang *et al.* (1989). In addition, numerous other epidemiological studies and animal studies also support the findings of Yang *et al.* (1989) (U.S. EPA, 1991a). In addition, the ATSDR (2003) chronic MRL for selenium compounds is also 5 µg/kg bw/day, and is based on the same endpoint (selenosis) and utilizes the same magnitude of uncertainty factor as the U.S. EPA oral RfD. Furthermore, Health Canada (1996) derived an oral PTDI (assuming a 70 kg individual) of 7.14 µg/kg bw/day, which is in close agreement with the U.S. EPA oral RfD. The National Academy of Science (IOM, 2000) has derived an acceptable Upper Limit (UL) based on the NOAEL of 800 µg/day based on Yang and Zhou (1994). Application of two-fold uncertainty factor results in an upper limit of approximately 5 µg/kg/day for adults. There was

no evidence of increased selenium toxicity for any age group (similar sensitivity for all ages) (IOM, 2000). IOM (2000) also derived an infant specific UL, based on a study by Brätter *et al*. (1991), of 47 µg or approximately 7 µg/kg/day for two through six-month-old infants. Health Canada has indicated that in 2005/2006, they will officially ULs as toxicity reference values for all essential elements (Health Canada, 2005, pers. comm. Roest and Petrovic) for contaminated sites human health risk assessments. ). In the derivation of the *Guideline for Use at Contaminated Sites in Ontario* (MOE, 1996b), MOE utilized an oral RfD of 5 µg/kg/day for selenium.

For the purposes of this risk assessment an oral RfD/TRV of 5.00 µg/kg/day was selected for selenium (IOM, 2000; Health Canada, 2005).

Cancer (Non-threshold) Effects

Selenium compounds (with the exception of selenium sulfide) are widely considered non-carcinogenic, therefore, no regulatory agencies were identified that developed health-based exposure limits based on carcinogenic endpoints. As selenium sulfide is typically not present in soils, foods or other environmental media to a significant extent (ATSDR, 2003), human environmental exposure to selenium sulfide would likely be negligible, relative to other forms of selenium.

***Inhalation Exposure Limits***

Non-cancer (Threshold) Effects

The U.S. EPA, ATSDR, and Health Canada have not developed inhalation exposure limits for selenium compounds. The Ontario Ministry of the Environment provides two health-based limits for selenium in air, a point-of-impingement limit and a 24-hour ambient air quality criterion of 20 and 10 µg/m$^3$, respectively (MOE, 2001). The California Environmental Protection Agency has developed a chronic Reference Exposure Limit of 20 µg/m$^3$, for effects on the alimentary, cardiovascular and nervous systems, based on route-to-route extrapolation from the Yang *et al*. (1989) study (OEHHA, 2001). The REL was derived by multiplying the U.S. EPA oral RfD of 5 µg/kg/day by an inhalation extrapolation factor of 3.5 µg/m$^3$ per mg/kg-day. Details of the origin of this factor were not provided in the supporting documentation from OEHHA (2001); however it can be obtained simply by dividing a default body weight of 70 kg by a default inhalation rate of 20 m$^3$/day. Route-to-route extrapolation assumes by default that a chemical is equally absorbed *via* both inhalation and oral routes and that the 'first pass' effect due to metabolism by the liver is not significant (OEHHA, 2001).

For the purposes of this risk assessment a chronic REL RfC of 20 µg/m$^3$ was selected (OEHHA, 2001).

Cancer (Non-threshold) Effects

As selenium compounds (with the exception of selenium sulfide) are widely considered non-carcinogenic, no regulatory agencies were identified that developed health-based exposure limits based on carcinogenic endpoints. Selenium sulfide is typically not present in soils, foods or other environmental media to any significant extent, human environmental exposure to selenium sulfide would likely be negligible (ATSDR, 2003).

***Dermal Exposure Limits***

No regulatory dermal exposure limits for selenium compounds were identified in the literature reviewed for the current assessment. Route-to-route extrapolation was used to derive an appropriate limit for the current assessment.

### 4.2.4 Bioavailability/Bioaccessibility

One of the most important factors in determining exposure of target tissues to a substance, and the body's ultimate response, is *bioavailability*. Bioavailability is the fraction of the total amount of a substance to which an organism has been exposed that successfully enters the blood stream. The bioavailability of a substance is dependent on the chemical form, the environmental medium, the route of exposure, physiological characteristics of the organism at time of exposure (*e.g.,* ingested substances may be absorbed to different extents depending on whether the stomach is full or empty) as well as the tissues/organs with which the substance must interact as it passes from the point of entry to target tissues.

When applying exposure limits, it is necessary to consider the bioavailability of each substance in the particular study from which the exposure limit is derived, to obtain reasonable estimates of the quantity of the chemical entering the body of study animals or subjects. This allows for the normalization of exposures with respect to exposure route, and comparison of the bioavailable doses to humans with the exposure limits determined from animal studies or human epidemiological data. It is inappropriate to convert exposure estimates to absorbed doses if toxicity values are based on administered doses. However, if an exposure estimate is adjusted for bioavailability then it must be compared to an exposure limit which is based on an absorbed, rather than an administered dose. Otherwise, the estimation of potential impacts would be incorrect and may underestimate exposure and risk depending on the particular circumstances. Since most exposure limits are based on administered doses, it is not appropriate to consider absolute bioavailability (fraction or percentage of an external dose which reaches the systemic circulation) in the assessment of exposures in most instances. A better measure may be that

of relative bioavailability which can be determined by comparing the extent of absorption among several routes of exposure, forms of the same substance, or vehicles of administration (such as food, soil, and water). Systemic absorption of substances will differ according to whether the dose was received *via* dermal contact, ingestion or by inhalation. Also, systemic absorption will differ depending on whether the substance is delivered in a solvent vehicle (water, soil, food, *etc.)*.

As discussed previously, for some substances, exposure limits are not available for all exposure routes of concern. In cases when (1) an exposure limit is available for some exposure routes but not for the exposure route of concern; and, (2) no other data (such as pharmacokinetics) are available, it may be necessary to extrapolate an exposure limit from one route to another. For example, it is common in human health risk assessment to assess the risks posed by dermal absorption of a substance based on the exposure limit established for oral exposure. The systemic dose absorbed dermally is scaled to the "equivalent" oral dose by correcting for the bioavailability of the dermally-applied chemical relative to an orally-administered dose.

The oral bioavailability of a substance is typically determined from absorption or excretion studies. The bioavailability, expressed as a percentage, is generally assumed to be 100% minus the percent of the ingested chemical excreted unchanged in the feces. In cases where only the fraction of chemical in the urine is reported, this fraction is selected as the minimum oral bioavailability with the maximum being 100%. In the absence of relevant data, this approach is considered to be reasonable, and to reflect the uncertainty in the oral bioavailability of the chemical.

The relative absorption difference between the oral and dermal routes of exposure can be expressed as a relative absorption factor ($RAF_{dermal}$), which has been described previously.

Typically, adjustments of exposure limits for bioavailability are considered for systemic effects (*i.e.*, following entry into and distribution by the bloodstream, as opposed to occurring at the site of entry [*e.g.*, lungs, skin, gut]) when:

- The exposure limit is based on a different route of exposure (*i.e.,* when the criterion is based on ingestion and the exposure routes of interest are inhalation or dermal exposure);
- The medium of administration in the study used to develop the exposure limit results in a different bioavailability than the exposure medium of interest (*e.g.,* ingestion in drinking water *versus* ingestion in soil); or,

- If the bioavailability of the chemical, based on the particular study animal/receptor, is different from that of the receptor upon which the exposure limit is based (*e.g.,* the exposure limit is based on a study using mice, the species of interest is human, and there are reported bioavailabilities for both mice and humans).

In these cases, adjustment for bioavailability may be important in determining appropriate toxicological criteria for use in comparing to route-specific exposures, as well as ensuring that comparisons are made either for internal ("bioavailability-adjusted") doses and limits relevant to the species or population being assessed, or route-specific doses and limits. It allows for normalization of exposures with respect to exposure route, the calculation of total exposures through all routes, and allows the bioavailable doses to humans to be compared with bioavailable doses determined from animal studies. In cases where the bioavailabilities for the route of the estimated exposure and the route considered in the toxicological criterion development are the same, the bioavailability adjustment is, in effect, cancelled out by use on both sides of the risk characterization equation.

When evaluating the health risks related to exposures to metals, such as the COC being evaluated in the Sudbury HHRA, an important aspect of a substance's bioavailability is the *bioaccessibility* exhibited by that substance. Bioaccessibility is the mass fraction of a substance that is converted to a soluble form under conditions of the external part of the membrane of interest. If one is evaluating bioaccessibility *via* the oral route, it is the fraction of substance that becomes solubilized within the gastrointestinal tract (*i.e.*, stomach and small intestine). In the case of dermal exposures, it is the fraction solubilized on the outside of the skin (*i.e.*, in sweat). To better characterize this fraction, a detailed site-specific *in vitro* bioaccessibility study was conducted to estimate the bioaccessibility of each of the COC present in soil and indoor dust media collected as part of the overall study. These site-specific oral bioaccessibility studies were used to help address the differences in oral bioavailability observed in these media *versus* the medium used in the study from which the toxicological criterion was derived. A summary of the bioaccessibility study is provided in Section 3.2, with the detailed bioaccessibility report in Appendix J of this Volume.

In addition to performing site-specific bioaccessibility studies, the scientific and regulatory literature was reviewed to identify bioavailabilities for each route of exposure evaluated in the HHRA, and where possible, values specific to species (*i.e.,* humans) and to the environmental media of concern (*e.g.,* soil, dust).

Table 4.29 provides the results of the bioaccessibility testing conducted for each of the COC in both the soil and dust test media.

**Table 4.29 Summary of Bioaccessibility Results for this Study**

| Chemical | Bioaccessibility (%) | |
|---|---|---|
| | Soil | Dust |
| Arsenic | 39 | 45 |
| Cobalt | 28 | 30 |
| Copper | 74 | 49 |
| Lead | 78 | 95 |
| Nickel | 44 | 31 |
| Selenium | 26 | 67 |

In order to use the bioaccessibility results effectively in the risk assessment, the relative absorption factor (RAF) should be used, where appropriate. The RAF corrects for the differential media/matrix to which the samples for the bioaccessibility evaluations are conducted (*i.e.*, soil) and the media used in the study that was used to derive the RfD. For example, the originating study for the RfD of nickel soluble salts is based on the ingestion of rat chow by mice. Therefore, a correction for the bioaccessibility of nickel in rat chow should be applied to all bioaccessibility data gathered for soil samples. The RAF can be determined and applied to the exposure estimates for potential human exposure scenarios. Spiked rat chow was subjected to the bioaccessibility assay and an overall bioaccessibility of 94.7% was observed. This was the used in the RAF determination for nickel. For other COC, study absorption factors of 100% were assumed.

The results of the bioaccessibility study indicates that as much as 78% of the lead present in GSA soils becomes solubilized (*i.e.*, is available for absorption) in the gastric phase of the study. Similary, 95% of the lead present in dust collected from the GSA becomes solubilized in the gastric phase of the bioaccessibility study. Drexler and Brattin (2007) have related relative *in vivo* bioavailability (RBA) and *in vitro* bioaccessibility (IVBA) estimates from a large dataset of lead-contaminated soils and wastes. A highly significant correlation coefficient between the two sets of data was found and the following linear regression equation relating the two derived:

$$\mathbf{RBA = 0.878 * IVBA - 0.028}$$

This equation allows an estimate of RBA when only IVBA is known. In the current study, the IVBA estimates for lead (78% for soil and 95% for dust) results in estimates for soil RBA of 66% and dust RBA of 83%. These values were utilized in the current assessment.

Based upon the results of the bioaccessibility testing, the following RAF values were selected for use in the current HHRA.

**Table 4.30 Summary of Relative Absorption Factors (RAF) for the HHRA**

| Chemical | Relative Absorption Factors (RAFs) | | | |
|---|---|---|---|---|
| | Oral | | Inhalation | Dermal [c] |
| Arsenic | Soil | 0.39 | 1 [b] | 0.03 |
| | Dust | 0.45 | | |
| Cobalt | Soil | 0.28 | 1 [b] | 0.001 |
| | Dust | 0.30 | | |
| Copper | Soil | 0.74 | 1 [b] | 0.003 |
| | Dust | 0.49 | | |
| Lead [a] | Soil | 0.66 | 1 [b] | 0.001 |
| | Dust | 0.83 | | |
| Nickel | Soil | 0.42 | 1 [b] | 0.001 |
| | Dust | 0.30 | | |
| Selenium | Soil | 0.26 | 1 [b] | 0.001 |
| | Dust | 0.67 | | |

[a] The RAF for lead in soil and dust have been adjusted based on the Drexler and Brattin (2007) regression equation.
[b] Assumes 100% of $PM_{10}$ size fraction assumed to be available
c All dermal RAF values from RAIS (2004).

Refer to Chapter 3 and the toxicological profiles for each COC in Appendix A for further background information on the proposed absorption factors.

## 4.3 Risk Characterization

The risk characterization step integrates the exposure and hazard assessments to provide a conservative estimate of human health risk for the receptors assessed in the various exposure scenarios. Potential risk is characterized through a comparison of the estimated or predicted exposures from all pathways (from the Exposure Assessment) with the identified exposure limits (from the Hazard Assessment) for all chemicals of potential concern.

For the COC which are thought to be non-carcinogens, this comparison is typically called the Hazard Quotient (HQ) and is calculated by dividing the predicted exposure level by the exposure limit (see equation below).

$$\text{Hazard Quotient (HQ)} = \frac{\text{Estimated Exposure (ug/kg/day)}}{\text{Exposure Limit (ug/kg/day)}}$$

The HQ value is used as an indicator to:

- Identify situations where the exposure received by human receptors under a specified set of conditions is greater than the maximum allowable concentration or dose (i.e., exposure limit); and,
- Estimate potential impacts on human health from exposures to mixtures of chemicals, if appropriate.

Risk characterization for chemicals with non-threshold-type dose responses (*i.e.,* carcinogens) consists of a calculation of the Cancer Risk Level (CRL), which is defined as the predicted upper bound risk of an individual in a population of a given size developing cancer over a lifetime. However, it should be noted that these upper bound risks are unlikely to be exceeded, and the true risk is likely to be less, and may even be zero.

The CRL is expressed as the prediction that one person per *n* people would develop cancer, where the magnitude of *n* reflects the risks to that population; for example, if the CRL is one person per 10, the predicted risks of any individual developing cancer would be higher than if the CRL is one per 1,000. The following equation provides the method whereby the CRL is calculated:

$$\text{Cancer Risk Level (CRL)} = \text{Estimated Lifetime Exposure} \times \text{Cancer Slope Factor } (q_1^*)$$

The resulting estimated cancer risk can then be compared to an acceptable risk level of cancer to determine if exposures to the assessed chemical pose an unacceptable health risk. In many jurisdictions, including Ontario, an incremental lifetime cancer risk (ILCR) level of one-in-one million is considered acceptable to regulatory authorities. The selection of an acceptable risk level is predominantly a policy-based, rather than a science-based, decision. An ILCR refers to the contribution that a facility or site makes to the total risk. In situations like Sudbury, it is difficult to tease out the actual incremental contributions that the facilities have made. In this case, Sudbury specific risks have been calculated and as such, an alternate acceptable risk level may be appropriate.

HQs and CRLs are used to express the potential adverse health effects from exposures to the selected chemicals for several reasons:

- To allow comparisons of potential adverse effects on health between chemicals and different exposure scenarios (*e.g.,* Typical Ontario *versus* site-specific conditions);
- To estimate potential adverse effects on health from exposures to mixtures of chemicals that act on similar biological systems (*e.g.,* All chemicals that cause liver toxicity, or kidney toxicity, or respiratory tract cancers); and,
- To simplify the presentation of the HHRA results so that the reader may have a clear understanding of these results, and an appreciation of their significance.

Some chemicals may act *via* multiple mechanisms; for chemicals of this nature both cancer (CRL) and non-cancer (HQ) risk estimates are calculated.

### 4.3.1 Evaluation and Interpretation of Hazard Quotients and Cancer Risk Levels

The information presented in this section applies to both deterministic and probabilistic approaches. When using the deterministic exposure analysis approach, HQs and CRLs are given as point estimate values. However, when one uses the probabilistic exposure analysis approach, the results of the human health risk assessment are expressed as a frequency distribution forecast or profile. The frequency distribution forecasts provide the full range of possible risk estimates for each chemical, receptor and scenario combination that is evaluated. For the probabilistic assessment, many of the parameters modelled incorporate a distribution of the expected range of values, rather than single point estimates. Consequently, the final results of the probabilistic distribution forecast enable a greater use of available information than assessments that consider only single, point estimate values. By incorporating a range of values for parameters, rather than a single value, the influence of variability on different exposure and

hazard parameters can be estimated and its significance evaluated. Generally, the focus of probabilistic human health risk assessment results is on the 95$^{th}$ percent upper confidence limit of the distribution forecast, although it is also important not to lose sight of the entire distribution forecast. This also facilitates comparison of frequency distribution forecasts between different scenarios (*e.g.,* study area *versus* typical Ontario).

The evaluation and interpretation of HQs and CRLs can be applied with greatest confidence to situations where comparisons are made between the HQs/CRLs of two or more independent exposure scenarios. From such comparisons, the incremental difference in the potential for occurrence of adverse health effects between the two or more different scenarios (*e.g*., study area *versus* typical Ontario) can be assessed with reasonable confidence since the same exposure and hazard assessment methodologies are used in addressing each situation. Most of the uncertainties in such comparative assessments are related to the ability to accurately estimate COC concentrations in the various environmental media that determine the different exposure pathways, and in the estimation of the toxicological criteria that exposure estimates are compared against.

**Hazard Quotients (HQ)**

Once HQ values have been determined for threshold chemicals (non-carcinogens), they are compared to a benchmark indicator of "safety", which is sometimes called the Critical Hazard Quotient (CHQ). In general, if the total chemical exposure from all pathways is equal to, or less than the exposure limit, then the HQ would be 1.0 or less, and no adverse health effects would be expected. Therefore, the benchmark of safety would be 1.0, assuming that estimates of exposure from all relevant exposure pathways are included.

However, for threshold chemicals, the exposure limits (or toxicological criterion) represent the level of total exposure, which would not result in adverse health effects, regardless of the source or pathways of exposure. As most risk assessments generally evaluate single or few sources of contamination and a limited number of exposure pathways, the selection of a CHQ value of 1.0 for threshold chemicals is not always appropriate. In an attempt to address this issue, the CCME (1996) considers that a substance has the potential to be present in all media, and assumes an allocation of 20% of the residual tolerable daily intake for each of the five major media (*i.e.,* air, water, soil, food, consumer products). Similarly, the MOE recommends apportioning 20% of the total exposure to any one pathway (MOEE, 1987), in the absence of information to the contrary. This means that the overall CHQ (*i.e.,* 1.0) must also be apportioned for the single source (*e.g.,* a contaminated site) under consideration. This yields a value of

0.20, which can be considered as the CHQ representing a situation in which no adverse health effects are likely to be associated with the estimated level of exposure for a given pathway. Therefore, if threshold chemicals are determined to have HQ values less than 0.20, exposure rates are considered to be less than 20% of the exposure limit (toxicological criterion), and no adverse health effects would be expected to occur in the receptors and scenarios evaluated in the risk assessment. If HQ values are greater than 0.2, the estimated exposure rates are considered to exceed 20% of the exposure limits, indicating the potential for adverse effects in sensitive individuals or in some of the exposure scenarios considered.

It should be noted however, that if the risk assessment included estimation of exposures to COC that are not associated with the study area under investigation, then it can be assumed that the risk assessment considers all significant sources of exposure, and that the total exposure of each receptor is being adequately accounted for. In this case, all significant sources of exposure were accounted for, with the exception of consumer products. The SARA Group conducted a detailed literature search and was unable to locate any information that indicated that consumer products would be a significant source of inorganic exposures (like the COC in the current study). As such, the SARA Group recommends the use of a CHQ value of 1.0 to represent an "acceptable level" of exposure, while recognizing there is some uncertainty with respect to the potential contribution of consumer products to an individual's EDI.

**Cancer Risk Levels (CRL)**

For non-threshold chemicals (*i.e.,* chemicals believed to act as carcinogens), the risk characterization is based on limiting ILCR to some level considered "negligible" or "acceptable". As previously discussed, the ILCR represents the predicted incremental risk of cancer over a lifetime to an individual member of a population of a given size, and is expressed as a risk level (*e.g.,* one person per *n*). Calculated ILCRs are compared to a benchmark risk level that is considered to be acceptable by the responsible regulatory agency in a given jurisdiction. In Ontario, the MOE specifies an acceptable ILCR of one-in-one million ($1x10^{-6}$). In other jurisdictions, negligible or *de minimis* cancer risk levels are generally considered to be in the range $1x10^{-4}$ to $1x10^{-7}$ (Health Canada, 2004a). An ILCR refers to the contribution that a facility or site makes to the total risk. In situations like Sudbury, it is diffult to tease out the actual incremental contributions that the facilities have made, since Sudbury is an urban environment with inputs other than the facilties. In this case, Sudbury specific risks have been calculated and as such, an alternate acceptable risk level, such as one-in-one million, may be appropriate. MOE has used an acceptable risk level of one-in-one hundred thousand in the derivation of ambient air criteria, as ambient air measurements cannot be attributed to a single facility and as such are not turly incremental in nature.

Where estimated risks (ILCRs for non-threshold acting chemicals) or risk indicators (HQs, for threshold-acting chemicals) are less than the acceptable level, it can be concluded that no observable adverse health effects would be expected to occur including sensitive subpopulations or groups, within the exposure scenarios considered in the HHRA. Risk estimates that are substantially less than the acceptable level are not considered to require further evaluation. In situations where risks are predicted to be within the same order of magnitude as the acceptable level, re-evaluation of certain model parameters (*e.g.,* chemical concentration estimates, exposure parameters, and toxicological criteria) is conducted before the potential risks to health are fully characterized. In these situations, consideration must be given to the possibility of adverse health effects, but a slight exceedence (or lack of exceedence) of the acceptable risk benchmarks do not typically indicate a high potential for risk. The methods and assumptions used in this HHRA are designed to be conservative (*i.e.,* health protective), and have a built-in tendency to overestimate, rather than underestimate, potential health risks. Thus, risk estimates that are within an order of magnitude of the acceptable risk benchmarks may reflect overestimation through the use of overly conservative assumptions and parameters (*e.g.,* overestimating exposures through use of maximum soil ingestion rates). In these cases, interpretation of the risk estimates may indicate that given the conservatism of the assessment, no adverse health effects would be expected despite the exceedance of the acceptable risk level or, that further assessment (*i.e.,* progression to a more detailed and specific risk assessment that could involve further data collection or probabilistic exposure analysis), or mitigative measures are warranted.

When predicted risks are substantially greater than the acceptable level (*i.e.,* more than 10-fold), the potential for adverse effects in sensitive individuals or in some of the exposure scenarios is suggested. Again, however, the re-evaluation of such HQs/ILCRs is extremely important since both the exposure estimation procedures and the toxicological criteria are based on a series of conservative assumptions that tend to overestimate exposures and risks. Often, a sensitivity analysis is conducted which facilitates the re-evaluation by focusing on the proportional contribution of various parameters to the final HQ/ILCR value. Once the major contributing model parameters have been identified, they can be re-evaluated to determine their impact on the resulting risk estimates and whether health risks have been under-estimated or over-estimated. Most often, the sensitivity analysis indicates that exposures and risks were overestimated. This occurs because a certain amount of over-estimation of risk is inherently built into the risk assessment process. For example, in cases where there is considerable uncertainty in the data such as the determination of toxicological criteria for cancer causing chemicals (*e.g.,* arsenic), a conservative dose-response extrapolation model is used to derive the toxicological criterion to ensure the protection of

human health. In probabilistic analyses, the estimates of potential adverse effects on human health at the upper end of the distribution forecast (*e.g.,* the 95$^{th}$ percentile upper confidence limit on the mean) represent the combination of numerical parameter values that occur infrequently based on the frequency distribution functions used for the various parameter values. Re-evaluation of the basis for these values at the upper end of the frequency distribution forecast must be conducted prior to recommending any remedial or other mitigative actions that would be based on these risk estimates. The outcome of this re-evaluation may include recommendations towards progressing to additional probabilistic analyses, additional data collection, or remedial action. Probabilistic analyses also allows risk managers to predict the effectiveness of different risk management activities by reducing exposure and risk profiles.

### 4.3.2 Consideration of Chemical Mixtures

Concurrent exposures to more than one chemical may result in interactions among toxicological effects; this may result in a combined toxicity which is equal to the sum of toxicities of the individual chemicals (additivity or independence), greater than the sum (synergism or potentiation) or less than the sum (antagonism). In general, toxicological interactions depend on the chemicals present, the levels of exposure to each, their mode of action and their concentrations. Most non-additive interactions can only be demonstrated at relatively high exposures, where clear adverse effects are observed. Such interactions have not been observed or quantified at the relatively low rates of exposure typical of those associated with most environmental situations (NAS, 1983; Krewski and Thomas, 1992).

The decision to evaluate individual substances separately was made in the case of the current HHRA. A detailed discussion of this topic is provided in Section 6.4 of this volume.

## 4.4 Risk Management Recommendations

If, after careful review and consideration of the factors described previously, the results of the risk characterization indicate that there may be unacceptable risks posed to some receptors of concern, then preliminary recommendations towards mitigation of those risks can be made. Risk management recommendations may suggest possible ways in which exposure pathways contributing significantly to overall exposure and risk can be limited or eliminated. For example, if contact with surface soils is driving risk, depending on the current and future uses of the land, it may be appropriate to simply put a layer of asphalt or clean fill over the contaminated soil, thereby preventing soil contact and mitigating the risk. Soil amendments, such as liming, can also be used to mitigate risks, in that they can modify the availability of chemicals in the soil. In some cases it may be necessary to remove contaminated media to mitigate risk. In cases where it is determined that risk management is necessary, risk management soil levels (SRMLs) are used to guide potential remediation activities. SRMLs are also referred to as risk management criteria (RMC) for intervention levels or premiminary remediation goals (PRGs) by some agencies.

The need to recommend a SRML is based on a number of key considerations including:

1. The nature, extent and duration of the risk and the uncertainties in how risks are estimated;
2. Evidence or lack of evidence of actual harm to health in the community; and
3. Outcomes of risk assessments in other communities with similar or higher levels of exposure.

There may also be legal, financial, political, and community concern-based issues that play a role in the establishment of suitable SRMLs and subsequent action that may be taken.

The SRML can be defined as the average concentration within an exposure unit (EU) that corresponds to an acceptable level of risk (U.S. EPA, 2001a). In other words, the SRML is the exposure point concentration (EPC) within a given exposure unit (EU) (*i.e.*, a COI) which would yield an acceptable level of risk. It is noted that the EPCs used to facilitate the long-term (or chronic) exposure assessment and subsequent approximation of hazard and risk were defined as the upper 95% confidence limit on the arithmetic mean (95% UCLM) from a specific or community of interest. Risks are based on a conservative approximation of the true (or population) mean of community-specific environmental media, and in essence assume that individuals move in a random fashion with their residential community. In reality, individuals do not move in a random fashion within their residential community, but rather exhibit some type of predictable spatial pattern in their movements. For example, many individuals will tend to

spend the majority of their time between home, work and/or school. If the SRML is defined as the EPC (*i.e.*, the 95% UCL on the arithmetic mean) in soil within a given community which yields an acceptable level of risk, then some residential properties will exceed the EPC. Depending on how the soil concentration data are distributed, it is plausible that the remediation of a number of highly impacted soils within the community could bring the overall EPC for that community below the SRML. If the property or site of concern was a single residential lot, it would be reasonable to assume that an individual would move in random fashion within his or her own residential property. The removal of a number of highly impacted zones to facilitate the reduction in the EPC for this single property may be a reasonable approach. However, because the exposure units of interest represent entire communities, in which individuals do not spatially move in a random fashion, the remediation of locally impacted zones to reduce the overall EPC for the community is not valid. The SRML values should be applied to individual residential properties, not necessarily the community as a whole. The result is that on a community wide basis, no unacceptable risk may be predicted while on a site specific basis, some properties may exceed the SRML for that community.

A variety of SRMLs can be derived for each of the COC depending on the statistic (mean, UCLM, RME, CTE, percentile value of a probabilistic distribution of risk) deemed appropriate for the protection of human health in the GSA. For the current assessment, the risk predictions from the RME receptor exposure scenario (*i.e.*, reasonable upper bound) were used to generate the SRML for lead (see Section 5.4 for a further discussion of this issue).

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